COMPARING THE SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE – DHAKA REGIME WITH PRITCHARD REGIME IN THE MANAGEMENT OF ECLAMPSIA

Dissertation submitted for

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APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled **"COMPARING THE SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE – DHAKA REGIME WITH PRITCHARD REGIME IN THE MANAGEMENT OF ECLAMPSIA"** submitted for **M.D BRANCH II OBSTETRICS AND GYNAECOLOGY**, The Tamilnadu Dr.MGR Medical University, Chennai April 2013 is a bonafide work done by **Dr.R.SASIKALA**, under my direct supervision and guidance in the **DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY, THANJAVUR MEDICAL COLLEGE, THANJAVUR** during her study period.

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DECLARATION

I **Dr.R.Sasikala** solemnly declare that the dissertation titled **"COMPARING THE SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE – DHAKA REGIME WITH PRITCHARD REGIME IN THE MANAGEMENT OF ECLAMPSIA"** is bonafide work done by me at Department of Obstetrics & Gynaecology, Thanjavur Medical College, Thanjavur under the guidance and supervision of my beloved Prof.Dr.S.Swaruparani, M.D.,D.G.O.,

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in the partial fulfillment of requirement for the award of M.D. degree Branch Obstetrics & Gynaecology degree examination to be held in April 2013.

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ABBREVATIONS

1.MgSO4	Magnesium sulphate
2.IV	Intravenous
3.IM	Intra muscular
4.IUGR	Intra Uterine Growth Retardation
5.NMDA	N –Methyl D-aspartate
6.PGE_1	ProstaglandinE_1
7.PGE_2	ProstaglandinE_2
8.$PGF_2\alpha$	Prostaglandin$F_2\alpha$ (Prostodin)
9.HELLP	Hemolysis, Elevated liver enzymes, Low platelets
10.PIGF	Platelet Inhibiting Growth Factor
11.VEGF	Vascular Endothelial Growth Factor
12.TGF	Transforming Growth Factor
13.NO	Nitrc Oxide

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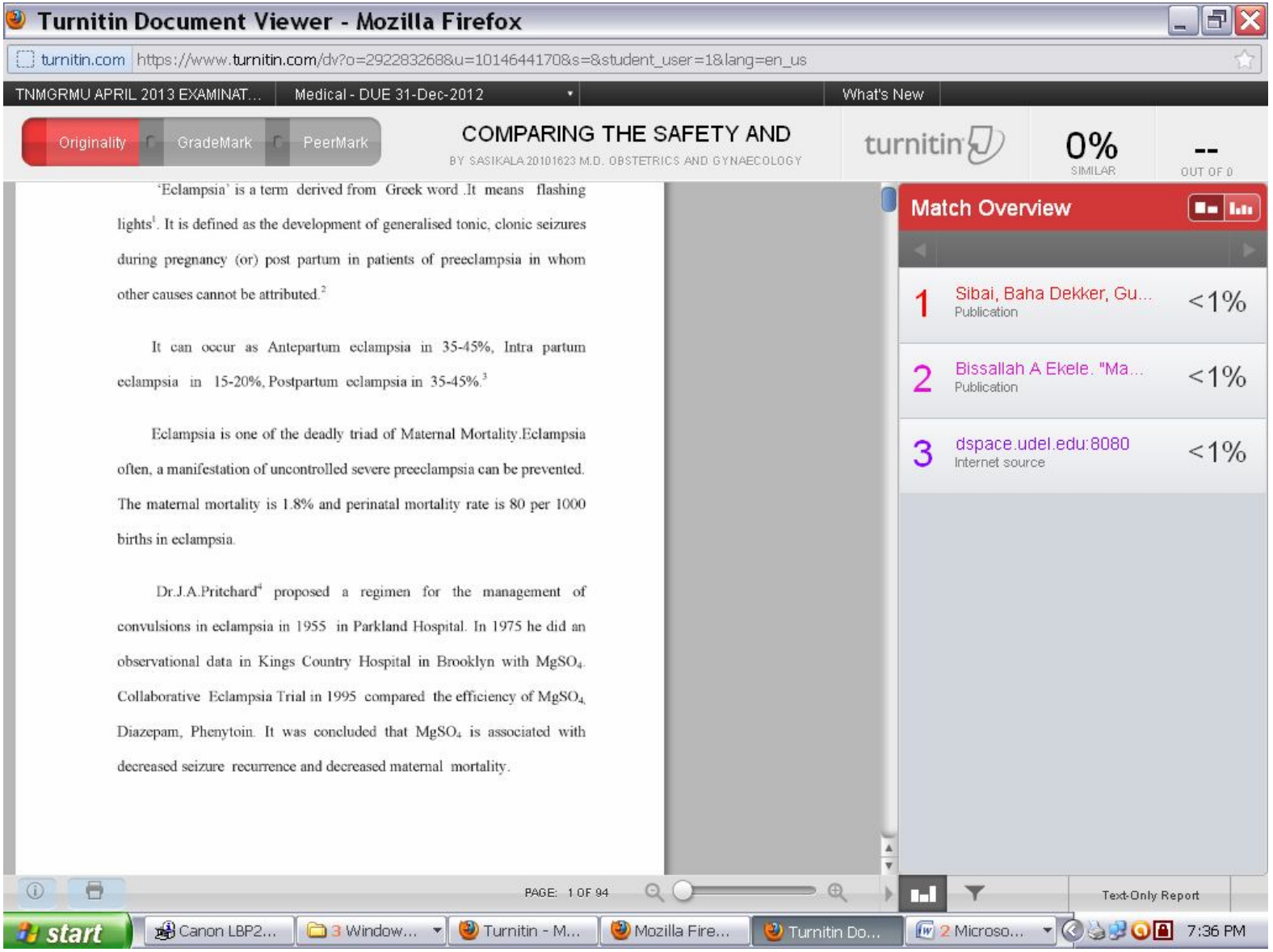
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'Eclampsia' is a term derived from Greek word .It means flashing lights[1]. It is defined as the development of generalised tonic, clonic seizures during pregnancy (or) post partum in patients of preeclampsia in whom other causes cannot be attributed.[2]
It can occur as Antepartum eclampsia in 35-45%, Intra partum eclampsia in 15-20%, Postpartum eclampsia in 35-45%.[3]
Eclampsia is one of the deadly triad of Maternal Mortality.Eclampsia often, a manifestation of uncontrolled severe preeclampsia can be prevented. The maternal mortality is 1.8% and perinatal mortality rate is 80 per 1000 births in eclampsia.
Dr.J.A.Pritchard[4] proposed a regimen for the management of convulsions in eclampsia in 1955 in Parkland Hospital. In 1975 he did an observational data in Kings Country Hospital in Brooklyn with $MgSO_4$. Collaborative Eclampsia Trial in 1995 compared the efficiency of $MgSO_4$, Diazepam, Phenytoin. It was concluded that $MgSO_4$ is associated with decreased seizure recurrence and decreased maternal mortality.
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ABSTRACT

COMPARING THE SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE – DHAKA REGIME WITH PRITCHARD REGIME IN THE MANAGEMENT OF ECLAMPSIA

AIMS

To study the safety and efficacy of low dose Magnesium Sulphate (Dhaka Regime) in Eclampsia and to compare the maternal and perinatal outcome in Dhaka and Pritchard Regimes.

METHODS

This is a Randomised control study done during Jan 2011 to Jan 2012 including 100 patients. 50 were treated under Pritchard Regimen in which a loading dose of 4 gm of 20% $MgSO_4$ IV and 10 gms of 50% $MgSO_4$ Im given. Maintenance dose was given as 5gm of 50% $MgSO_4$ repeated at 4 hour intervals till 24 hours after the last fit or 24 hours after the delivery of fetus whichever is later.

50 Eclamptic patients were treated under Dhaka Regime in which loading dose of 4 gm of 20% $MgSO_4$ IV and 6gms of 50% $MgSO_4$ Im given and repeated at 4 hourly intervals in which 2.5gm of 50 % $MgSO_4$ given as maintenance dose. Recurrence of fits, maternal and perinatal outcome in both groups were compared.

RESULTS AND ANALYSIS

Recurrence of fits was noticed in 2% each in Dhaka and Pritchard regime. The maternal mortality in this study is 2% each in Dhaka and Pritchard regimen. The cause of death in both patients were due to complications of Eclampsia. The perinatal mortality in Dhaka regimen is 20% and perinatal mortality in Pritchard regimen is 30%

CONCLUSION

The low dose efficiency of $MgSO_4$ – Dhaka regime is sufficient for the management of eclampsia and it is equivalent to the standard Pritchard regimen.

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INTRODUCTION

'Eclampsia' is a term derived from Greek word .It means flashing lights[1]. It is defined as the development of generalised tonic, clonic seizures during pregnancy (or) post partum in patients of preeclampsia in whom other causes cannot be attributed.[2]

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Dr.J.A.Pritchard[4] proposed a regimen for the management of convulsions in eclampsia in 1955 in Parkland Hospital. In 1975 he did an observational data in Kings Country Hospital in Brooklyn with $MgSO_4$. Collaborative Eclampsia Trial in 1995 compared the efficiency of $MgSO_4$, Diazepam, Phenytoin. It was concluded that $MgSO_4$ is associated with decreased seizure recurrence and decreased maternal mortality.

In 1998 March to June, in Dhaka,[5] capital of Bangladesh, a prospective study including 65 patients of eclampsia treated with low dose Magnesium sulphate was conducted. It was concluded that the low dose $MgSO_4$ which is nearly half that of Pritchard was sufficient to control the convulsion effectively in Eclampsia.

AIM

- The safety and efficacy of low dose Magnesium Sulphate regime in the management of convulsion in Antepartum / Intrapartum / Postpartum eclampsia patients.

- This study also compares the maternal and perinatal outcome in patients treated with Pritchard and Dhaka regimen.

REVIEW OF LITERATURE

DEFINITION:

Eclampsia is defined as the onset of convulsions or coma in women who have either gestational hypertension or pre eclampsia. Unfortunately in 15% cases, hypertension and proteinuria are absent. However, when seizures develop in a pregnant women without a known history of seizure disorder, Eclampsia should be the diagnosis until proved otherwise.

INCIDENCE:

In developed countries 1 in 2000 to 1 in 4000 deliveries is complicated by eclampsia . In developing countries 1 in 100 to 1 in 1700 deliveries is complicated by eclampsia.

TYPES OF ECLAMPSIA:

Antepartum eclampsia --convulsion occurring before labor(35-45%)

Intrapartum eclampsia – convulsion during labor(15-20%)

Post partum eclampsia – convulsion after labor(35-45%)

ETIOLOGY :

Though many theories have been proposed for the etiology, It is still unclear. Eclampsia being an extreme degree of pre eclampsia, the biochemical changes and histo pathological changes are the same for both, except, that it is most pronounced in eclampsia.

Mechanisms currently proposed to explain the courses of eclampsia are culmination of maternal, placental and fetal factors.

They are

1) Implantation of placenta with abnormal trophoblast invasion of uterine vessels. There is incomplete trophoblastic invasion of spiral arterioles.

   Madzali and associates (2009)[6] proved that severity of hypertension is directly proportional to the magnitude of defective trophoblastic invasion.

2) Maladaptive Immunological tolerance between maternal ,placenta and fetal tissues. There is immune tolerance from mother towards paternally derived placental and fetal antigens. This is proved by the occurrence of preeclampsia more in primigravida, high occurrence of preeclampsia in molar pregnancy, 30 to 40% incidence of preeclampsia in Trisomy 13.

3) Maladaptation to cardiovascular and inflammatory changes of normal pregnancy

4) Genetic factors

5) Nutritional factors :

It was reported by Zhang[7] and associates (2002) that if ascorbic acid intake is less than 85 mg it is associated with pre eclampsia.

Lots of fruits intake is associated with low BP as shown by John[8] and co workers in(2006).

Villar[9] and associates (2002) proved that calcium supplementation has no beneficial role in the prevention of pre eclampsia. But supplementation of calcium reduces the perinatal mortality.

PATHOGENESIS

1. Vasospasm:

In 1918 Volhard put forward the concept of vasospasm based on the observation in small vessels of nail bed, fundus, and bulbar conjunctiva. Vasospasm cause increased resistance and subsequent hypertension.

In 2002 Wang[10] et al demonstrated endothelial junctional protein disruption. Endothelial disintegrity causes interstitial leakage through which blood platelets and fibrinogen are deposited subendothelially.

Suzuki et al[11] (2003) explained the ultra structural changes in sub endothelial region of resistance arteries in pre eclamptic women. With reduced blood flow, there is ischemia of surrounding tissue leading to necrosis, haemorrhage and other end organ damage.

2. Endothelial cell activation:

Anticoagulant property of intact endothelium is reversed on subendothelial activation and increase the sensitivity to vasopressor.

In 2008 Grundmann[12] and associates have found that circulating endothelial cell levels (CEC) are significantly elevated four fold in peripheral blood of pre eclamptic mothers.

3. Increased Pressor Response:

In 1961 Abdul–karim[13] Assali reported that normally pregnant women have refractoriness to infused vasopressors.

But in preeclamptic women, they have increased response to infused Nor adrenaline, Angiotensin II and it was reported by Raab& coworkers in 1956.

In 1974 Gant[14] & colleagues noticed that this refractoriness develop several weeks before the onset of hypertension.

4. Prostaglandins:

Endothelial prostaglandins responsible for vascular refractoriness is diminished (PGI_2) .Similarly ThromboxaneA2 secretion by platelets is increased. Thus Prostacyclin : TX A_2 ratio is decreased.

These ultimately results in enhanced sensitivity to pressor agents and thereby cause vasoconstriction.

The above changes are evidenced in pre eclamptic women as early as 22 weeks of gestational age and the same was proved by Chavarria [15] & co workers 2003.

5. Nitric oxide

Nitric oxide derived from L- arginine by endothelial cells is a potent vasodilator.

Myat[16] et al in 1992 showed that Nitric oxide maintains the normal low pressure, vasodilator state characteristic of fetoplacental perfusion. Preeclampsia is associated with decreased endothelial nitricoxide synthase expression thus increasing inactivation of nitric oxide.

6. Endothelins:

Endothelin ET 1 – produced by human endothelium a potent vasoconstrictor is increased in normotensive in pregnant woman. In the preeclamptic woman, endothelins are much more increased.

Sagsoz[17] and kucukozkan in the year 2003 noticed that on administration of Magnesium sulphate, ET_1 concentration is reduced.

7. Angiogenic causes:

Karumanchi[18] and colleagues in 2009 found that in women who are prone to develop pre eclampsia produce minimum of 2 Anti Angiogenic peptides in maternal serum .They are

- Soluble FMS- like tyrosine kinase 1 .(SFlt -1) :

  are elevated reducing PIGF,VEGF thereby causing endothelial dysfunction. Maynard &associates 2003 has shown SFlt-1 will start rising many months before the clinical presentation of pre eclampsia.

- Soluble endoglin:

  Seng inhibits endoglin thereby prevents the binding of TGF β to the receptors in endothelium that causes NO thereby vasodilatation. Its level also starts rising months before clinical presentation of pre eclampsia as shown by Levine[19] & coworkers in 2006.

Riskfactors

1. Parity:

Eclampsia affects more young and nulli parous women.Whereas chronic hypertension with superimposed pre eclampsia affects multiparous women.

In 2009, Sibai[20] and Cunningham reported that there is 3 to 10% incidence of preeclampsia in nulliparous women.

2 .Genetic predisposition:

Pre eclampsia is a multifactorial, polygenic disease. Ward and Lindheimer (2009)[21] noticed that the risk for developing preeclampsia is 20 to 40% in the daughters. 11 to 37% in their sisters.

3. Environmental.

4. Seasonal influences

Occurs in winter and rainy season when there is increased humidity.

5. Socio economic.

6. BMI:

The risk of pre eclampsia is directly proportional to weight gain

In BMI > 35 kg/m2 the incidence is 13.3%

In BMI -20kg/m2 the incidence was only 4.3%

Con de Agudelo[22] &Belizan (2000) added other risk factors like

- Multifetal gestation
- Obesity
- Maternal age more than 35 years.

Getahun[23] & colleagues (2007) found that the incidence of preeclampsia is lower in subsequent pregnancies in woman with normal blood pressure in their pregnancy.

PATHOPHYSIOLOGY OF ECLAMPSIA

As formulated by Borzychowski[24] 2006, and Redman[25] 2009 the theory of two stage Disorder of pre eclampsia shows

Stage I: is preclinical: There is faulty trophoblastic vascular remodelling of uterine arteries resulting in placental hypoxia.

Stage II: placental factors release into maternal circulation resulting in systemic inflammatory response and endothelial activation.

Preeclampsia is more likely to develop in women who are for the first time exposed to chorionic villi in pre existing renal or cardiovascular disease and genetically prone women.

There is an imbalance in autoregulation of cerebrovascular system resulting in intense vasospasm (or) vasodilatation.

If the response to hypertension is vasospasm, there is

- ischemia,
- cytotoxic edema,
- tissue infarction.

As Schwartz[26] pointed out if the severe hypertension exceeds the normal regulatory capacity there is forceful vasodilatation causing extravasation of plasma and RBC through endothelial lining leading to vasogenic edema.

Meldrum[27] in 2002 reported that seizures are associated with excess release of excitatory neurotransmitters like Glutamate, burst of action potentials, large depolarization of network neurons.

In woman who died due to eclampsia, their autopsy findings revealed

- micro infarcts in cortical matter and white matter of brain
- cerebral edema
- cerebral parenchymal bleeding and
- vascular lesions more in Occipital lobe.

PRE MONITORY SYMPTOMS OF ECLAMPSIA:

Head ache:

It is due to hyperperfusion of brain especially Occipital lobe .since the anterior part of brain is well supplied by sympathetic innervations,

Occipital lobe is much affected . 50 to 75% eclamptic mothers has preceding headache which is not relieved by analgesics. Headache is mild to severe, constant or intermittent and improves after initiating MgSo4 therapy. The headache is described as throbbing type.

Visual disturbances:

20 to 30% have disturbances in vision before onset of eclamptic seizures. The different visual disturbances are diplopia, blurred vision, scotoma. They are relieved on lowering the Blood pressure or after $MgSo_4$ therapy. The symptoms are due to spasm of retinal arterioles, edema and retinal ischemia.

1 to 3 % of eclamptic cases have reversible blindness arise from 3 potential areas.

Visual cortex of brain

Lateral geniculate nuclei

Retina : retinal ischemia,infarction,detachment

It takes about 1 week for the blindness to get corrected after the delivery of fetus.

Changes in the mental status:

Ranges from lethargy, confusion,blurred vision to coma. It occurs due to cerebral edema.

Right upper quadrant pain (or) epigastric pain:

Due to stretching of Glisson's capsule due to edema or sub capsular haemorrhage, hepato cellular ischemia, necrosis.

Vomiting

Clinical stages of Eclampsia.

There are 4 clinical phases in an attack of eclampsia.

1. Premonitory stage:

The patient loses her consciousness. It is associated with twitching of facial muscles. It lasts for a period of less than 30 seconds.

2. Tonic stage:

It is associated with a spasmodic contraction of the entire body. It lasts for a period of 15 to 30 seconds.The whole body becomes rigid.arms are flexed.hands are clenched.

3 Clonic stage

It is associated with alternate contraction and relaxation of voluntary muscles. It lasts for approximately 1 minute. Tongue bite due to violent action on jaws. Then the intensity of the movement of muscles is reduced. The diaphragm becomes fixed during the seizures and there is no breathing.

Froth occurs in the mouth. After stertorous breathing the normal breathing recurs.

4 Phase of Coma

The movements stops.Time duration is variable**.**

DIFFERENTIAL DIAGNOSIS:

- Epilepsy
- Meningitis
- Encephalitis
- Tumour in Brain
- Cysticercosis
- Ruptured cerebral aneurysm in late pregnancy and puerperium.
- Cerebral malaria
- Hysteria

COMPLICATIONS OF ECLAMPSIA

MATERNAL COMPLICATIONS

1. Abruption 10%
2. neurological sequelae -7%
3. Aspiration pneumonitis -7%

4. Pulmonary edema -5%
5. Acute Renal failure -4%
6. HELLP syndrome 4%
7. Cardiac arrest 4%
8. Maternal death 1%
9. Maternl Injuries
    - Tongue bite
    - External injuries -bruise,fractures
    - Asphyxia due to swollen tongue occluding glottis.
10. Hyperpyrexia

FETAL COMPLICATIONS :

- IUGR
- Prematurity
- Asphyxia .

The perinatal mortality and morbidity is as high as 30-50%.

Fetal bradycardia persist after a fits.It may persist for 3 to 5 minutes only.

MANAGEMENT OF ECLAMPSIA

The principles in Management of eclampsia are

1) Control of convulsion with Inj.$MgSO_4$
2) Anti hypertensive medication
3) Avoidance of diuretics and limitation of intravenous fluids since pre eclamptic patients have high hemoconcentration.
4) Termination of pregnancy to achieve a cure.

SUPPORTIVE TREATMENT:

It includes care of the patient in a quiet, noise free room .

Position of the patient should be in lateral decubitus.

Continuous monitoring of the patient's general condition

- Pulse rate
- Blood pressure
- Respiratory rate
- Oxygen saturation.
- Input/ output chart should be maintained.

Airway, Mouth gag should be kept ready. Suction apparatus should be in the room for quick suctioning.

- Tongue bite is prevented by a soft mouth gag in between teeth.

- Urinary catheter should be inserted for monitoring urine output.
- All the blood investigations like Clotting Time,
    - Clot Retraction time,
    - Renal Function test,
    - Liver Function Test,
    - Complete Blood Count,
    - Peripheral Smear are done.

Thus complications like HELLP, DIC, Renal failure etc. are diagnosed. Cardio Vascular System, Respiratory System should be thoroughly examined

Antibiotics should be given.

One to one nursing care should be given.

CONTROL OF CONVULSIONS

In the year 1955 Pritchard presented a paper regarding the management of eclampsia with magnesium sulphate. In 1990, the randomized trials of management of eclampsia were published.

In 2002, MAGPIE[28] trial was conducted in about 10000 preeclamptic patients. Patients were randomly selected. They were divided into two groups and treated with $MgSO_4$ in one group and another with placebo .

It was found that in patients treated with $MgSO_4$ had significantly 58% lower risk for eclampsia than the placebo group. 1.9% had eclampsia in placebo group.But it was only 0.8% seizures in $MgSO_4$ group.

Before $MgSo_4$ various anticonvulsants were used. The different regimens are

1. Menon Regimen(1961)
2. Lean Regimen
3. Phenytoin

MENON REGIMEN:

This includes IV administration of 25mg Chlorpromazine & 100mg Pethidine in 20ml of 5% glucose and IM administration of 50mg Chlorpromazine & 25 mg Promethazine. The IM Injection of Chlorpromazine and Promethazine are repeated at 4 hourly intervals for 24 hrs after the last fit. Menon also used Lytic cocktail for 1448 eclamptic patients.

There was 2.2% Maternal Mortality Rate.

Side Effects were maternal and fetal respiratory depression.

Antidote used was Naloxone.

DIAZEPAM (LEAN REGIMEN)

This includes Intravenous administration of 10mg diazepam over 2mts followed by IV infusion of 40 mg in 500 ml NS for 24 hours. Thus by this diazepam infusion the patient is made sedated but arousable.

Maximum dose is 100 mg in 24 hours.

The side effect of diazepam is respiratory depression in mother and lethargy and apnoea of new born.Babies had intractable hypothermia and were floppy.

No antidote was available,

The maternal mortality was 5% in this regimen.

In 1990 Crowther compared the use of diazepam and $MgSO_4$. It was concluded that Recurrent eclampsia occurred in 7% in Diazepam treated patients. Whereas it was 5 % with $MgSO_4$ treated patients.

In Collaborative Eclampsia Trial, thrice the number of cases had recurrent fits when compared to $MgSO_4$. The relative risk was 0.33. In 2006 Royal College of Obstetricians and Gynaecologists restricted the use of Midazolam or Lorazepam to single dose. This is due to increased maternal mortality rate.

PHENYTOIN :

Blitz first prepared phenytoin. It was used as an anti convulsant drug in 1938.

Parenteral :

Dosage of phenytoin is 10mg/kg by slow IV bolus followed by 5mg / kg 2 hrs later.

Oral :

It is followed by oral dosage of 200mg twice daily for 48 hrs after delivery. Phenytoin is non sedative. The therapeutic serum levels of phenytoin is 40 to 100 micro mol /litre.

Adverse effects are

- Hypotension
- Cardiac arrhythmias
- Thrombophlebitis in the site of injection.

A comparative study involving phenytoin and $MgSO_4$ was studied in Lalla Ded Medical College at Srinagar ,Kashmir was conducted in the years 2004 to 2006.It was concluded that 24% recurrent eclamptic seizures were there. Such patients were switched over to $MgSO_4$. And no seizure recurred

thereafter. Thus $MgSO_4$ is an ideal anticonvulsant drug in the control of eclamptic seizures.

Sawney et al reported recurrence of seizure in 40 % cases with phenytoin and it was only 8% with MgSO4 in a group of 25 patients. Also Phenytoin was studied for the prophylaxis of eclampsia in patients with severe preeclampsia by Lucas in the year 1995 .In that 0.9% had eclampsia with phenytoin. There was no seizure in patients with $MgSO_4$.

In Collaborative Eclampsia Trial conducted in 1995 the recurrence of seizure was twice in phenytoin group when compared to $MgSO_4$. The maternal mortality was also twice that of the patients treated with $MgSO_4$.The relative risk was 0.5.

MAGNESIUM SULPHATE

The various Magnesium sulphate regimens were

1. Pritchard Regimen
2. Zuspan Regimen
3. Sibai Regimen
4. Padhar Regimen
5. Dhaka Regime
6. Sokkotto Regimen

In Parkland hospital in the year 1955, Pritchard started $MgSO_4$ treatment regimen which consist both IV & IM of $MgSO_4$

PRITCHARD REGIMEN

LOADING DOSE

Initially as soon as the patient is received after the eclamptic fits she is given intravenous administration of 4 gm of 20% $MgSO_4$ at a rate not exceeding 1g/mt . And Intramuscular administration of 5 gm of 50% $MgSo_4$ in each buttock as deep Intra muscular injection.

MAINTENANCE DOSE

5 gram of 50% $MgSO_4$ is given as deep intramuscular injection on alternate buttocks every 4 hours. $MgSO_4$ continued as IM maintenance dose for 24 hours after delivery (or) 24 hrs after the last fit whichever is later.

The injection is given with a 3 inch long, 20 gauge needle.

If there is recurrence of convulsion within 15 mts, 2gm of 20% $MgSO_4$ is given slow intravenously at a rate not exceeding 1gm /minute.

The following parameters are noted every 4 hours.

1. Knee jerk should be present
2. Respiratory rate should be more than 16/mt
3. Urine output should be atleast 30ml / hr

Pritchard's regimen though widely used, He suggested that women with low BMI should be given low dose $MgSO_4$.

In (1962) Flower[29] et al adjusted doses according to body weight. In 2003 Sardesai[30] suman et al studied low dose $MgSO_4$ in Indian women. He reported 90% control of eclampsia and safety and efficacy of low dose $MgSO_4$ inferred. In Dhaka regimen there was 98% control of eclampsia.

ZUSPAN REGIME (1964)

Loading dose:

Administration of 4gm of 20% MgSO4 by an infusion pump over 5 to 10 minutes.

Maintenance dose:

This is followed by 20% $MgSO_4$ at the rate of 1-2gm/hour by controlled infusion pump for 24 hrs after the delivery of fetus.

PADHAR REGIME[31]

Loading dose :

IV administration of 6 gm of 50% $MgSO_4$

Maintenance dose:

IM administration of 4 gm of 20% $MgSO_4$.

It was a Prospective study conducted in Padhar hospital, India. 95 eclampsia cases were studied.The result was that only one woman had recurrent fits. She was given 5 gm as IM maintenance.

SOKOTTO REGIME[32]

(Ultra Short)

Dose :

IV administration of 4 grams of 20% $MgSO_4$ and intramuscular administration of 10 gms of 50% $MgSO_4$.

At Sokkotto, capital city of Nigeria, a study was conducted in the year july 2007 to june 2008 to study the efficacy of the ultra short regimen in antepartum, intrapartum, postpartum eclamptic patients .

There was 7.4% of recurrent seizures within 4 hours of loading dose in this study. Out of 121 patients, 12 mothers died. Thus case fatality rate was 9.9% in that study. Thus it was concluded that the efficacy of Sokkoto's regimen was 92.6% in that study.

SIBAI REGIME[33]

Loading dose:

IV administration of 6 gm of 20% MgSO4

Maintenance dose:

IV infusion of 20% MgSO4 for 24 hours after the fit.

DHAKA REGIME (1998) :

Loading dose :

4 gm of 20% $MgSO_4$ IV at a rate not exceeding 1 gm per minute. IM 6 gm of 50% $MgSO_4$ as deep IM, 3gm in each buttock.

Maintenance dose:

2.5gm of 50% $MgSo_4$ IM on alternate buttock every 4 hours. This prospective study was done in in Dhaka Medical college hospital in March to June 1998.Out of 65 eclamptic patients only one developed recurrent fits that occurred 3 hours after the loading dose. It was treated with diazepam and intramuscular maintenance dose. It was concluded that half the dose will be enough to control the fits.

MAGNESIUM SULPHATE

- The chemical formula of $MgSO_4$ is $MgSO_4. 7H_2O$.
- It is also called as Epsom salt.
- 1 gm of $MgSO_4$ has 98mg of elemental Magnesium.
- The molecular weight of $MgSO_4$ is 24.3.
- Ph is 6.0.(5.5 to 7.0) .

- The Osmolarity of 50% $MgSO_4$ is 4.06 mosm/ml.
- It should be stored in room temperature. (15 to 30 deg C)

Horn first used $MgSO_4$ intrathecally to avoid eclamptic fits prophylactically in the year 1906.

PLASMA LEVELS AND DISTRIBUTION

40% of plasma magnesium is protein bound after administration. The remaining 60% of magnesium diffuse into extravascular compartment like the extracellular space. It crosses the placenta, into the fetus and liquor. The volume of distribution reaches constant levels in the 4^{th} hour after administration. There are 2 phases in the pharmacokinetic profile of $MgSO_4$. They are Rapid Distribution phase and Slow phase of Elimination.

EXCRETION

Magnesium sulphate which is given in is cleared by renal excretion only. 50% of the dose is excreted after 4 hours and 90% of the total dose is excreted with in a period of 24 hrs. Glomerular filtration rate can be evaluated by the estimation of serum creatinine.

MECHANISM OF ACTION:

Mechanism of action of $MgSO_4$ which are proposed are

CENTRAL ACTION:

- Decreases the pre synaptic release of neurotransmitter Glutamate,an excitatory neurotransmitter.
- Blocks the Glutaminergic N- methyl D–aspartate NMDA receptors in Hippocampus which has a low seizure threshold.

PERIPHERAL ACTION:

- Adenosine action is potentiated
- Improves the calcium buffering action in mitochondria.
- Blocks the calcium entry through voltage gated channels.It was reported by Arango[34] and mejaMantilla in 2006 .

Thus $MgSo_4$ has a central anticonvulsant action as well as peripheral action on neuro muscular junction.

- $MgSo_4$ is a potent vasodilator in mainly cerebro vasculature. Therefore it reverses the vaso constriction which is the main cause for preeclampsia and thereby eclampsia.

In 1986 Cotton[35] and Colleagues found that there is a fall in Mean Arterial Pressure and 13% rise in cardiac index .

Other effects of $MgSO_4$

1. **Vascular effects:**

   Vasodilator in vascular beds resulting in Flushing,Head ache

2. **Uterine effects :**

Transient decrease in the myometrial activity immediately during and after the loading dose. Hence it is used in preterm labor.

FETAL EFFECTS OF $MgSO_4$

Neonatal depression is directly related to hypermagnesemia. It also affects fetal heart rate especially beat to beat variability. Hence biophysical profile is not useful at time of $MgSO_4$ adminstration.It is also reported that $MgSO_4$ prevents the neonates from developing cerebral palsy.

Nelson[36] and Grether (1995) conducted randomized trials and assessed the neuro protctive effects.

Doyle[37] and associates (2009) conducted five randomised trials including 6145 infants and concluded that gross motor dysfunction was reduced in the babies of mothers treated with $MgSO_4$.

SERUM MAGNESIUM LEVEL

- Normal serum mg level is 1-2 meq/L
- Therapeutic level of serum magnesium:-

Usually eclamptic fits are controlled when the plasma magnesium level is 4 to 7 meq/L

ADVANTAGES OF $MgSO_4$

Magnesium Sulphate is

- very efficient
- fastly acting
- wide safety margin
- not toxic to fetus and mother
- easy for administration and for monitoring
- effective antidote
- low cost
- cause less sedation.

TOXIC EFFECTS OF MAGNESIUM AND MONITORING OF SERUM MAGNESIUM

- When serum Mg level is 9-10 meq/L the patellar reflex becomes absent. The $MgSO_4$ regime is withheld. Serum Magnesium can be estimated if there is loss of patellar reflex, but it is very rarely needed since there is a wider margin of safety.

Duley at al[38] (1995) showed that there is no need to measure the Serum Magnesium level. Serum magnesium measurement is used only for the patients who develop magnesium toxicity symptoms.It is also measured when serum creatinine is more than 1 mg/dl

- When serum Mg level reaches above10meq/L respiration becomes weakened due to curare effect.
- When Mg level in plasma reaches above 12 meq/L respiratory paralysis and respiratory arrest follow.

There is no adverse effect on cardiac function.

Antidote:

1g of 10% Calcium gluconate or calcium chloride intravenously over 15 minutes. Along with this magnesium sulphate is stopped to reverse the toxic effect of respiratory depression.

For severe respiratory depression or respiratory arrest mechanical ventilatory support becomes necessary.

$MgSO_4$ IN RENAL FAILURE:

Even if the patient already has renal failure, the loading dose of Magnesium Sulphate can be safely given. This is because after distribution

of Magnesium, a loading dose of $MgSO_4$ achieves the desired therapeutic level and the maintenance dose (infusion dose) maintains the steady level. Thus only maintenance dose should be reduced in cases of renal failure.

American[39] College of Obstetricians and Gynecologists (2002) a ; Royal College[40] of Obstetricians and Gynecologists(2006) has recommended that it is not needed to measure the plasma magnesium level in all cases routinely.

DRUG INTERACTIONS WITH MAGNESIUM SULPHATE

Agent	Effect of $MgSO_4$	Recommendations
Neuromuscular blocking agents- depolarising & non depolarizing (succinyl choline)	Activity is increased	Dosage of neuro muscular blockers should be reduced.
Depressant drugs of central nervous system. General anaesthetics- opioids, barbiturates	Additive effect of CNS depression	Needs reduction of dose of the CNS depressant drugs
Nifedepine	Hypotension	Concurrent administration can be avoided

ANTI HYPERTENSIVE DRUGS

CLASS	DRUG	AVAILABILITY FORM	STARTING DOSE	MAXIMUM DOSE	SIDE EFFECTS
β Adrenargic blockers					
	Labetolol	50,100mg	100mg TDS	2400 mg	Edema, Fatigue
Alpha Blocker	Prazosin	1mg	1mg BD	20mg	Drowsiness Dizziness
Centrally Acting	➢ Alpha methyl dopa	250mg	TDS	2g	Postural hypotension drowsiness depression dry mouth,
Calcium Channel blockers	Nifedepine	5mg 10 mg	10mg TDS	120mg	Headache fatigue

HYDRALAZINE:

It is an arterial vasodilator

American College of Obstetricians and Gynaecologists (2002) has recommended the following dose.

DOSE :Initially 5 mg IV .It is repeated at interval of 15 to 20 minutes as 5 or 10 mg till the diastolic BP becomes 90 to100 mmHg. A maximum of 5 IV doses can be repeated .

Onset of action can be as rapid as 10 minutes. BP thus lowered saves the patient from developing Intra cerebral haemorrhage.

If the dose is given more than recommended ,it results in decreased utero placental perfusion leading to fetal heart rate decelerations.

Side effects are palpitation, tachycardia.

LABETOLOL:

It is an α and β blocker. The National High BP Education Programme working group (2000) and American College of Obstetricians and Gynaecologists in (2002)recommended the following dose.

- 20 mg IV bolus.

If it did not decrease the BP within 10 minutes, the dose can be doubled every ten minutes upto a maximum of 220 mg dose per episode .

Oral dose : 50 mg BD

Side effects are hypotension, bradycardia.

Vigil De Gracia[41] and associates (2007) reported that there was no difference in maternal and perinatal outcome in between patients treated with Hydralazine and Labetolol.

NIFEDIPINE :

It is a dihydropyridine derivative-- Calcium Channel Blocker

Dose recommended by National High BP Education programme and Royal College of Obstetricians and Gynaecologists in 2006 is

Initially 10 mg orally and it is repeated every 30 minutes.maximum dose is 200mg.

Side effect:

head ache

ankle edema

flushing

palpitation

hypotension

tachycardia

ROLE OF DIURETICS:

Diuretics causes intravascular volume depletion and thereby decreases utero placental blood flow. Hence Zeeman[42] (2009) has recommended the limited usage of diuretics only in pulmonary edema.

OBSTETRICAL MANAGEMENT OF ECLAMPSIA

Royal College of Obstetricians and Gynecologists Guidelines (2005) says that Termination of pregnancy is the definitive treatment of eclampsia.

The mode of Delivery should be vaginal in order to avoid maternal risk due to caesarean section. Per vaginal examination is done to assess the Bishop score of cervix and if the cervix is favorable, induction of labour is done using prostaglandin (PGE_2) gel (or) augmentation of labour done using oxytocin administration. Thus PGE_2 and Oxytocin are used for the successful vaginal delivery. Blood pressure should monitored and anti hypertensive drug should be used whenever necessary throughout labour and after labour too.

Alanis & associates[43](2008) reported that labour ensues spontaneously following an eclamptic fit, or can be successful even if the pregnancy is remote from term. This is because in antepartum eclampsia the uterine contractions will increase in frequency and intensity. If the seizure delivery interval is more, when waiting for vaginal delivery there may be serious morbidity in the postpartum period for the patient. Hence the seizure to delivery interval has a significant role.

The caesarean section (or) hysterotomy is done in the following situations:

1. Obstetric Indication
2. Unfavorable cervix so that vaginal delivery is impossible within 6 to 8 hours of occurrence of first episode of eclamptic fits.
3. All deeply unconscious patients and if the delivery is not imminent.
4. All uncooperative patients due to postictal confusional state
5. Fetal distress

MANAGEMENT OF LABOUR

The first stage of labor is monitored for uterine contractions. Partograph maintained. I/O Chart maintained.

The second stage of labour should be cut short and elective forceps delivery can be used.

The third stage of labour should be treated actively to prevent Post Partum Haemorrhage. Since the eclamptic patients have decreased blood volume when compared to normal pregnant these patients cannot withstand postpartum haemorrhage.

So PPH is prevented by

1. Inj. oxytocin 10 units IM after the delivery of anterior shoulder (or)
2. Inj.Prostaglandin $PGF_2\alpha$ 250μg as intramuscular injection (or)
3. Tab.Misoprostol(PGE1 analogue) 600 to 800μg kept per rectally.

Methyl erogometrine is contraindicated since it causes increase in blood pressure.

Blood transfusion is given for the required cases.

POST PARTUM CARE:

After the delivery of baby blood pressure, urine output, general condition of the patient should be monitored continuously for a period of 48hrs of delivery.Limitation of IV fluids 75 ml/hr was done because of the high hemoconcenteration.

In recent years, in developed countries due to intensive prenatal care, ante partum eclampsia is reduced and the incidence is more in postpartum eclampsia.

Chames[44] & colleagues (2002) showed that an incidence of 20% of the total elcampsia cases can be postpartum and hence the postnatal

period should be carefully monitored. Anti hypertensive drugs can be tapered off gradually as the blood pressure is controlled .

- First sign in the postpartum period to get well is the better urine output.
- Edema disappears after one week after of delivery.
- BP takes two weeks to become normal
- Proteinuria disappears after one week.

Reversible cerebral vasoconstriction syndrome

Some patients continues to have high BP, recurrent fits, neurological sequalae. It is seen in patients with severe vasospasm which causes ischemic and infarct areas in brain.

MATERIALS AND METHODS

This study was conducted at Raja Mirasudhar Hospital, Thanjavur in the Department of Obstetrics & Gynaecology during the period of January 2011 to January 2012 and 100 eclampsia patients including antepartum eclampsia, intrapartum eclampsia, post partum eclampsia were included for the purpose of this study. Magnesium Sulphate was used for the management of eclampsia. 50 eclamptic patients were treated with Pritchard regimen and other 50 with Dhaka regimen.

INCLUSION CRITERIA

All eclamptic woman antepartum, intrapartum, postpartum patients were included in the study irrespective of their age, gestational age, parity and status of booking.

EXCLUSION CRITERIA

- Patients who have already received Magnesium sulphate outside.
- Patients with known epilepsy
- Patients with known Heart Block, Renal Failure
- Onset of seizures more than 72 hrs after delivery in post partum eclampsia cases

Totally about 134 eclampsia cases were admitted in the period between January 2011 to 2012. Of which 34 patients were excluded according to exclusion criteria.

STUDY DESIGN

Randomized control Trial .

GROUP P:

Treatment of 50 patients randomly with Pritchard's regimen of Magnesium Sulphate.

GROUP D:

Treatment of 50 patients randomly with Dhaka regimen of Magnesium Sulphate.

HISTORY

History was elicited from the patient and her attendants if she was brought in a postictal state or unconscious state. Then the history was confirmed when the patient regained her consciousness. History regarding her age, parity, booking status, gestational age, number of eclamptic fits before admitting here, whether she was a known case of pregnancy induced hypertension, whether she is on anti hypertensive drugs, presence

of edema if so how long, existence of imminent symptoms like head ache, vomiting, blurring of vision were all elicited thoroughly. BMI was calculated from her old records. Any known history of Epilepsy, Renal failure, Heart block were also elicited in a detailed manner.

CLINICAL EXAMINATION

General examination of the patient from head to foot was made.

If the patient was admitted in a state of throwing fits suction of air way done.And airway maintained .IV line started and $MgSO_4$ administered according to the regimen. During this time bed side rails are elevated. A soft padded mouth gag is inserted in between teeth to prevent gag reflux.

On General examination, the following were noted.

- the state of consciousness of the patient ,
- the presence of anaemia
- degree of pedal edema,
- presence of facial edema,edema in hands
- Pulse Rate, Blood Pressure, Temperature, Respiratory Rate all were measured.
- Cardiovascular system, Respiratory system were examined thoroughly.

- IV line was started and blood samples sent for Renal Function Test, Liver Function Test, Prothrombin time, Complete blood count .
- Fundus examination was done by ophthalmologist
- The patient was catheterized with foley's indwelling catheter and urine output noticed immediately and monitored every hour. Urine sample was sent for estimation of proteinuria
- Continuous monitoring of oxygen saturation, pulse rate , Blood pressure were recorded.
- Knee jerk, Respiratory rate were noticed every 4 hours .

OBSTETRIC EXAMINATION

Per abdomen examination of the uterine fundus height, presence of contractions noted.viability of the fetus assured by fetal heart rate.

Per vaginal examination done under strict aseptic precautions. favourabilty of the cevix assessed by Bishop score.

MANAGEMENT OF ECLAMPTIC FITS:

STUDY GROUP:

DHAKA REGIMEN:

CONTROL GROUP:

PRITCHARD REGIMEN

Here patients were randomly selected and treated in both Regimens.

MANAGEMENT OF BLOOD PRESSURE WITH ANTI HYPERTENSIVE DRUGS

Hypertension is treated with antihypertensive drugs like Tab.Nifedepine 10mg and IV Labetolol.

OBSTETRIC MANAGEMENT:-

A detailed obstetric examination was made .Mode of delivery was decided according to the Bishop scoring ,Gestational age and the viability of fetus.

Labour was induced with prostaglandin PGE2 gel. The patients who came in active phase of labour were accelerated with amniotomy if membranes present and oxytocin infusion.If membranes absent oxytocin

infusion initiated. During labor uterine contractions and FHR monitored.partograph maintained.

Ceasarean section (or) Hysterotomy was performed in patients with unfavorable cervix, obstetric indication and for failed induction.

Perinatal outcome was also observed with the APGAR score.birth weight noted.The babies were followed up till the day of discharge.

After the delivery, the eclampsia patients were watched vigilantly,MgSO4 continued till 24 hours after the delivery of fetus or 24 hours after the last fit. Only when they were stabilised, they were shifted to post operative ward or post natal ward and observed there and the patients were followed up till the date of discharge. On the time of discharge she was advised to review in postnatal OP even if she maintained normal BP . Counselling regarding future pregnancy was given . Regular antenatal visits were insisted to diagnose pre eclampsia at an early stage for low dose aspirin. This is because recurrent preeclampsia is 3.4% in subsequent pregnancy and recurrent gestational hypertension is 25%.

Postnatally patients were monitored BP and treated with Tab. Atenolol, Tab.Nifedepine, Tab. Enalapril.

OUTCOME MEASURES:

Recurrence of fits was considered as the primary outcome measure after starting the patients on both Magnesium sulphate regime. The outcome of mother as maternal morbidity and mortality were compared. Perinatal outcome were also compared in both regimens.

RESULTS AND ANALYSIS

TABLE : 1

DISTRIBUTION OF AGE

Age Group	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Below 20yrs	12	24.0%	13	26.0%
21 to 25yrs	24	48.0%	23	46.0%
26 to 30yrs	9	18.0%	14	28.0%
31yrs & above	5	10.0%	0	0%
Total	50	100.00%	50	100.00%
Mean	23.80		23.26	

In the present study 25 patients were below 20 years.Between 21 to 25 years there were 47 patients.23 patients were between 26 to 30 years.The mean age of the patients in Dhaka regimen was 23.8 .The mean age in Pritchard patients was 23.26. In both Dhaka and Pritchard regime , the age of the patients does not differ significantly.

FIG. 1

DISTRIBUTION OF AGE

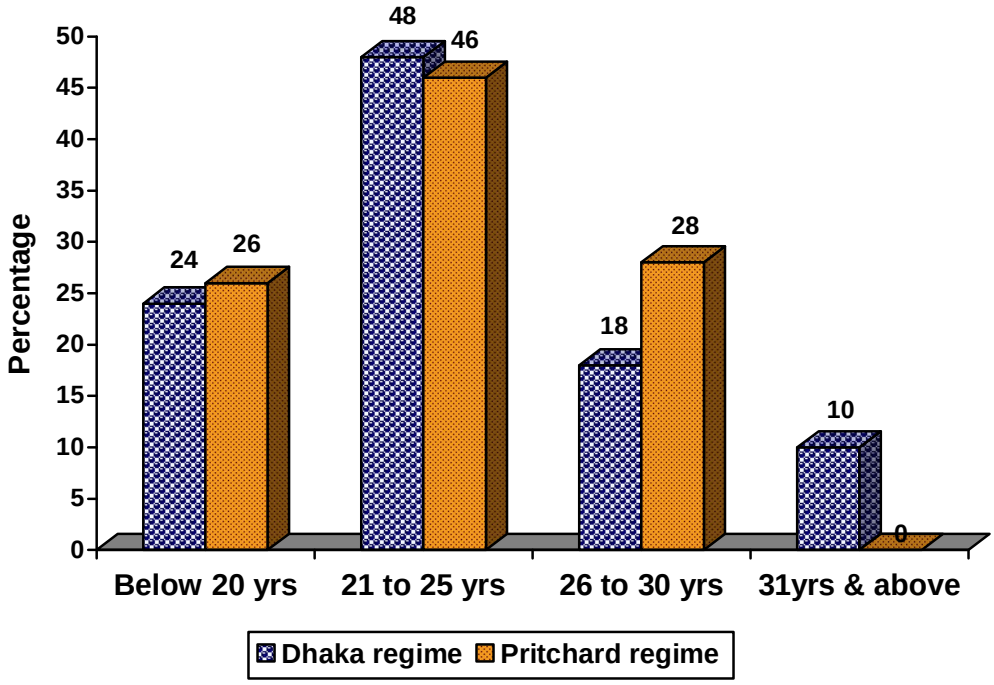

TABLE : 2

DISTRIBUTION OF PARITY

Parity	Dhaka regime		Pritchard regime	
	(n=50)	**(100%)**	**(n=50)**	**(100%)**
Primi	30	60.0%	31	62.0%
Multi	20	40.0%	19	38.0%
Total	50	100.00%	50	100.00%

Total primi in the present study is 61.

31 primi (62%) and 19 multi (38%)were treated under Pritchard regimen. Under Dhaka regimen 30 primi (60%) and 20 multi (40%)were treated.

FIG. 2

DISTRIBUTION OF PARITY

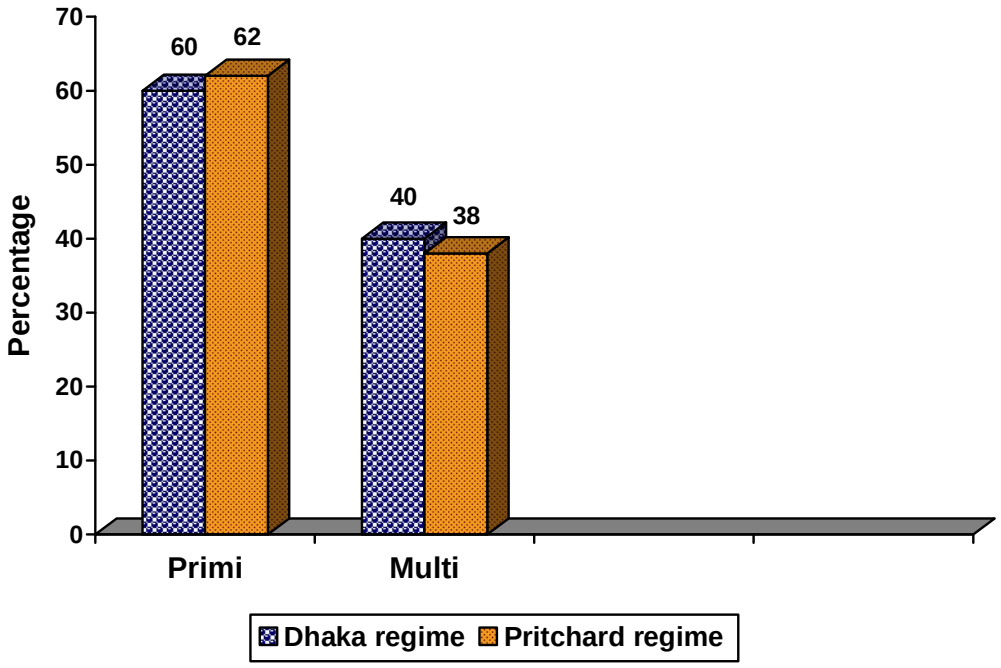

TABLE : 3

STATUS OF BOOKING

Age Group	Dhaka regime		Pritchard regime	
	(n=50)	**(100%)**	**(n=50)**	**(100%)**
Booked	30	60.0%	30	60.0%
Unbooked	20	40.0%	20	40.0%
Total	50	100.00%	50	100.00%

Total number of booked patients were 60.30 each in Dhaka and Pritchard regime. Unbooked patients were 20 each in both the regimens.

FIG. 3

STATUS OF BOOKING

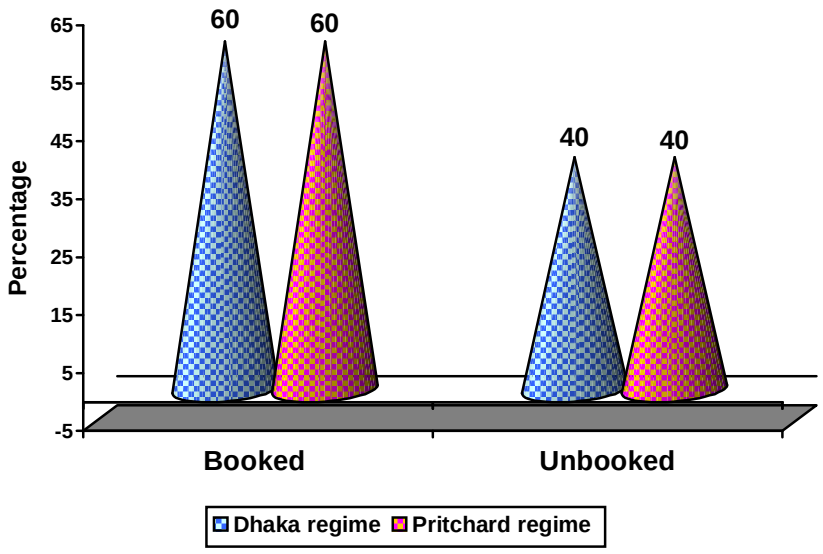

TABLE : 4

GESTATIONAL AGE

Gestational Age (weeks)	**Dhaka regime**		**Pritchard regime**	
	(n=50)	**(100%)**	**(n=50)**	**(100%)**
Below 28	6	12.0%	4	8.0%
28 to 37	26	52.0%	29	58.0%
More 37	18	36.0%	17	34.0%
Total	50	100.00%	50	100.00%
Mean	34.91		35.06	
P Value	0.856 (insignificant)			

In the study group 26 patients were between 28 to 37 weeks.18 were more than 37weeks.6 patients were below 28 weeks.

In the Pritchard group 29 patients were between 28 to 37 weeks. 17 patients were more than 37 weeks.4 were below 28 weeks.

The mean gestational age for Dhaka regimen group is 34.91 weeks. The mean age for Pritchard regimen was 35.06 weeks. The p value is 0. 856.

FIG : 4

GESTATIONAL AGE

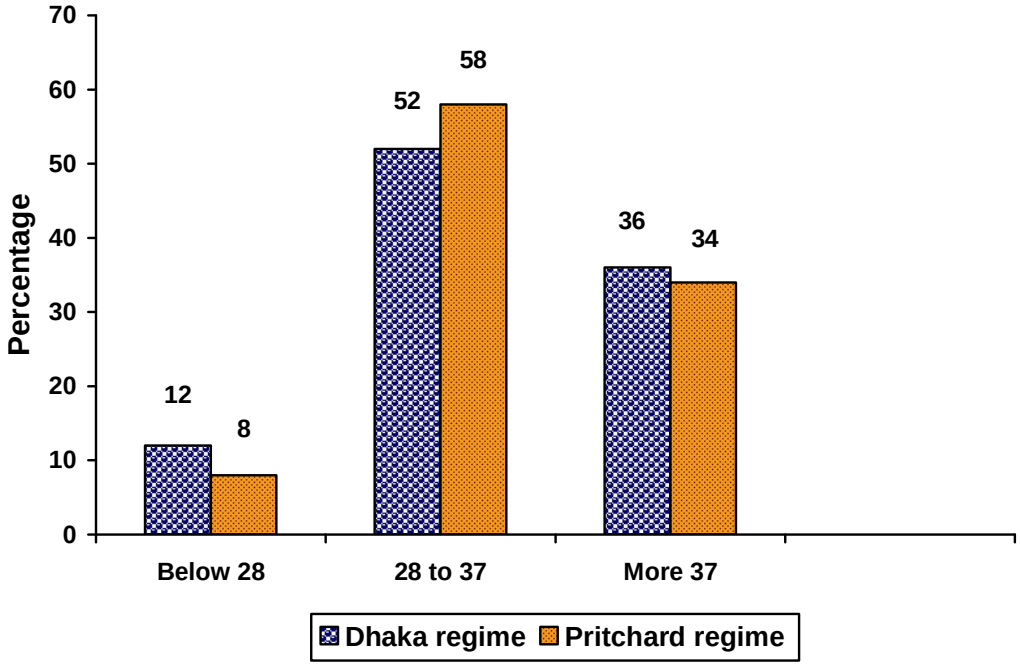

TABLE : 5

DISTRIBUTION OF BODY MASS INDEX

BMI	Dhaka regime		Pritchard regime	
	(n=50)	**(100%)**	**(n=50)**	**(100%)**
Low (<19.8)	5	10.0%	0	0%
Normal (19.8 to 26)	41	82.0%	45	90.0%
High (26 to 29)	3	6%	4	8.0%
Obese (>29)	1	2%	1	2.0%
Total	50	100.00%	50	100.00%

In Dhaka regimen group, 41 patients were with normal BMI.5 patients were with low BMI. In Pritchard regimen group 1 obese, 4 with high BMI and 45 with normal BMI were treated. The p value is 0.03.

FIG : 5

DISTRIBUTION OF BODY MASS INDEX

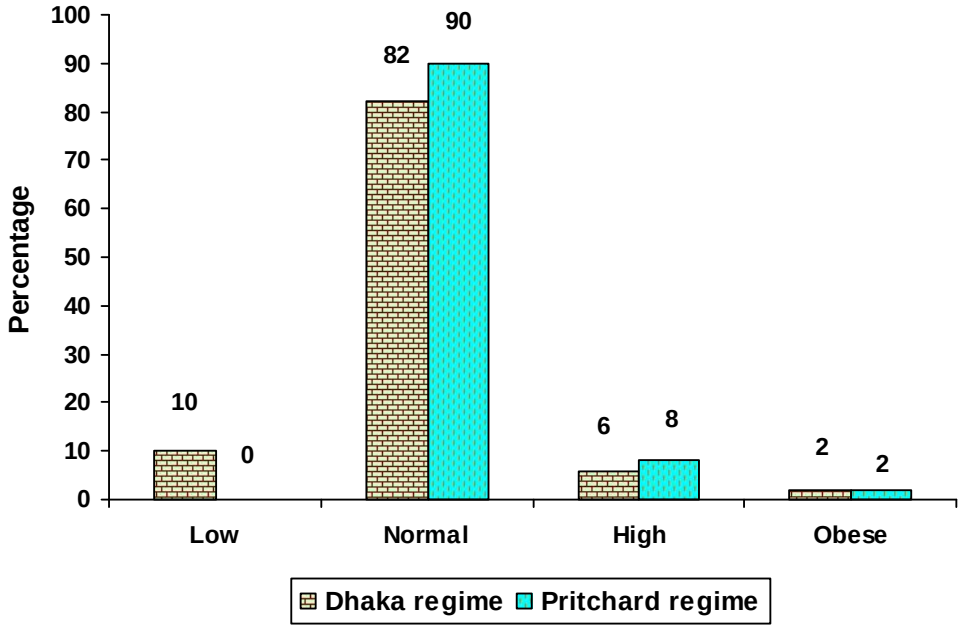

TABLE : 6

STATE OF CONSCIOUSNESS

STATE OF CONSCIOUSNESS	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Conscious	44	88.0%	44	88.0%
Post ictal confusion	4	8.0%	4	8.0%
Un consious	2	4.0%	2	4.0%
Total	50	100.00%	50	100.00%

In patients treated under Pritchard regimen 44 were admitted in conscious state.4 were in post ictal confusion and 2 were brought in an unconscious state. In patients treated under Dhaka regimen 44 were conscious,4 were in postictal confusional state and 2 were unconscious.

FIG : 6

STATE OF CONSCIOUSNESS

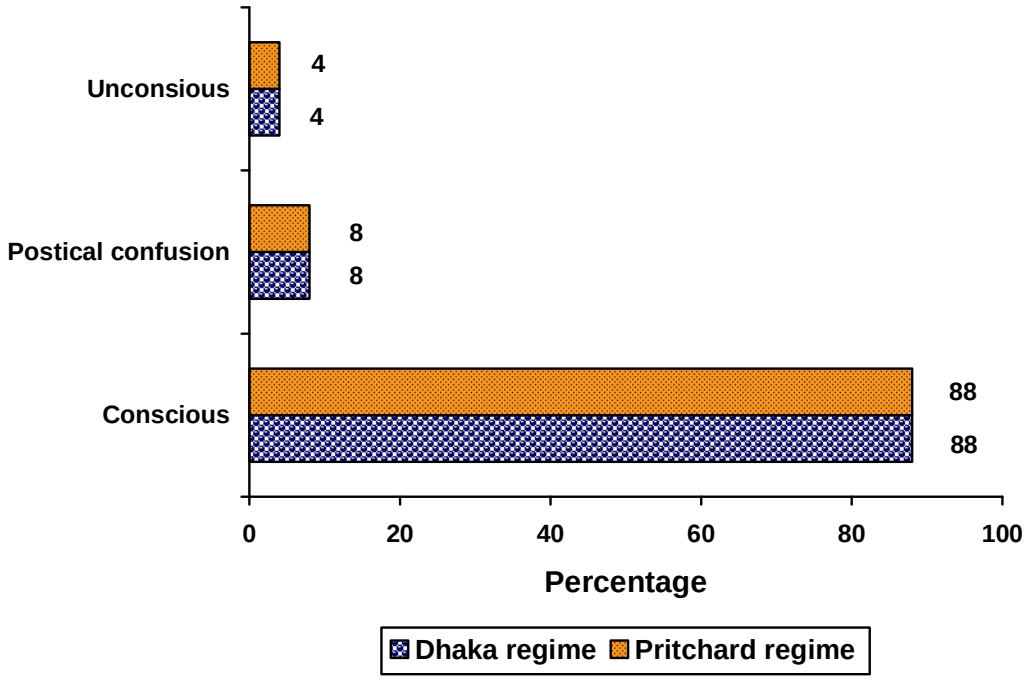

TABLE : 7

TYPES OF ECLAMPSIA

TYPES OF ECLAMPSIA	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Antepartum	40	80.0%	43	86.0%
Intrapartum	4	8.0%	1	2.0%
Postpartum	6	12.0%	6	12.0%
Total	50	100.00%	50	100.00%

Among the 83 antepartum eclampsia patients, 40 were treated under Dhaka regimen. 43 patients were treated under Pritchard regimen. 6 postpartum eclampsia patients were treated under Dhaka regimen as well as under Pritchard regimen. 1 intrapartum eclampsia patient was treated with Pritchard regimen. 4intrapartum eclampsia cases were treated under Dhaka regimen.

FIG : 7

TYPES OF ECLAMPSIA

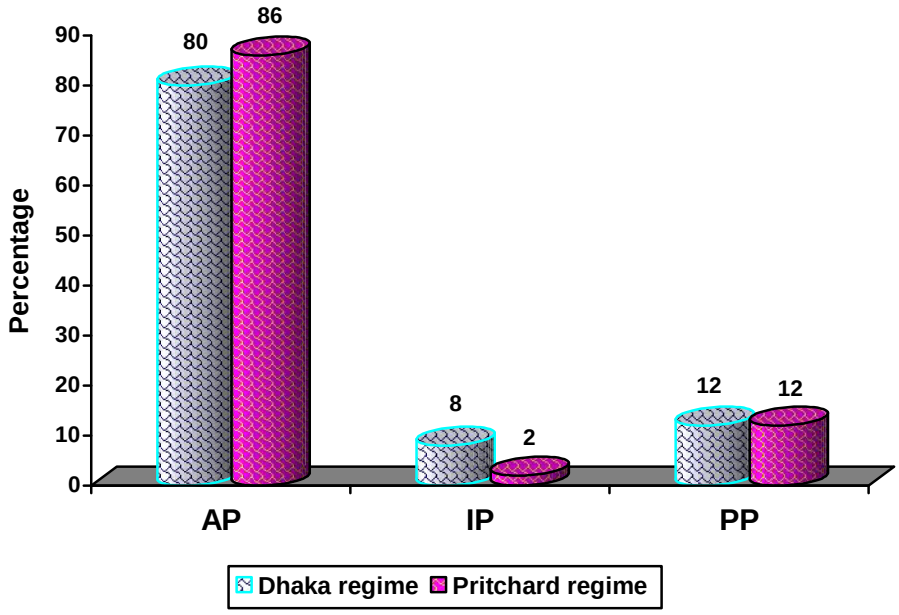

TABLE : 8

SYSTOLIC BLOOD PRESSURE

SYSTOLIC BLOOD PRESSURE	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Below 120	2	4.0%	4	8.0%
121 to 140	14	28.0%	9	18.0%
141 to 160	6	12.0%	15	30.0%
161 & above	28	56.0%	22	44.0%
Total	50	100.00%	50	100.00%
Mean	154.56		149.60	

In Dhaka regimen 28 patients had systolic BP above 161mmHg ,16 patients had below 140 mmHg . Among them 2 were below 120 mmHg. The mean systolic BP was 154.56mmHg in patients treated under Dhaka regimen. In patients treated under Pritchard 22 had systolic BP above 161 mmHg. 13 were below 140 mmHg. Within that 4 had BP below 120 mmHg . The mean systolic BP was149.60 mmHg. The p value is 0.681 and it is insignificant. Thus 12% had BP even below 120 mmHg.

FIG : 8

SYSTOLIC BLOOD PRESSURE

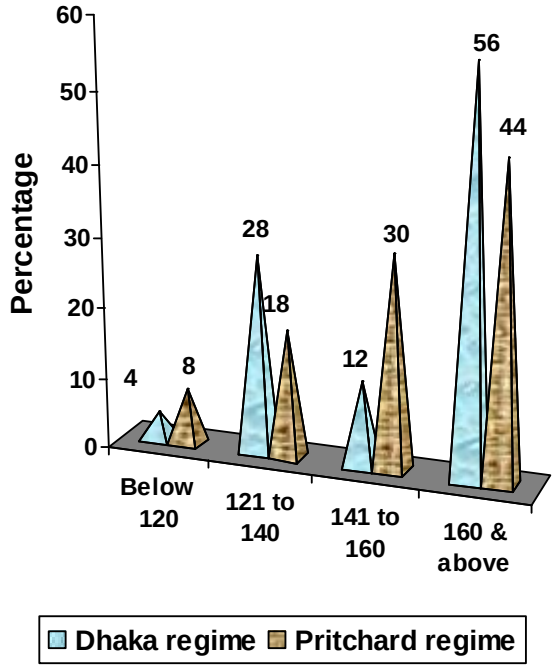

TABLE :9

DIASTOLIC BLOOD PRESSURE

DIASTOLIC BLOOD PRESSURE	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Below 90	3	6.0%	6	12.0%
90 - 99	11	22.0%	9	18.0%
100 - 109	18	36.0%	16	32.0%
110 & more	18	36.0%	19	38.0%
Total	50	100.00%	50	100.00%
Mean	100.00		98.80	

18 Patients under Dhaka regimen had more than 110 mmHg diastolic BP. Another 18 had diastolic BP in the range of 100 -109 mmHg.11 had diastolic BP in the range of 90 -99 mmHg.3 patients had diastolic BP below 90 mmHg. The mean value is 100mmHg.

In Pritchard regime 19 patients had diastolic BP more than 110 mmHg.16 patients had 100-109 mmHg. 9 had 90-99 mmHg.6 had normal BP. The mean value is 98.8 mmHg.

FIG : 9

DIASTOLIC BLOOD PRESSURE

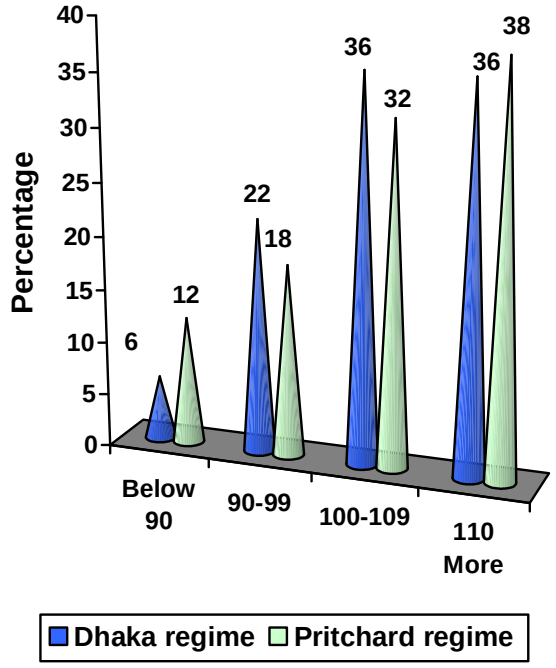

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TABLE : 10

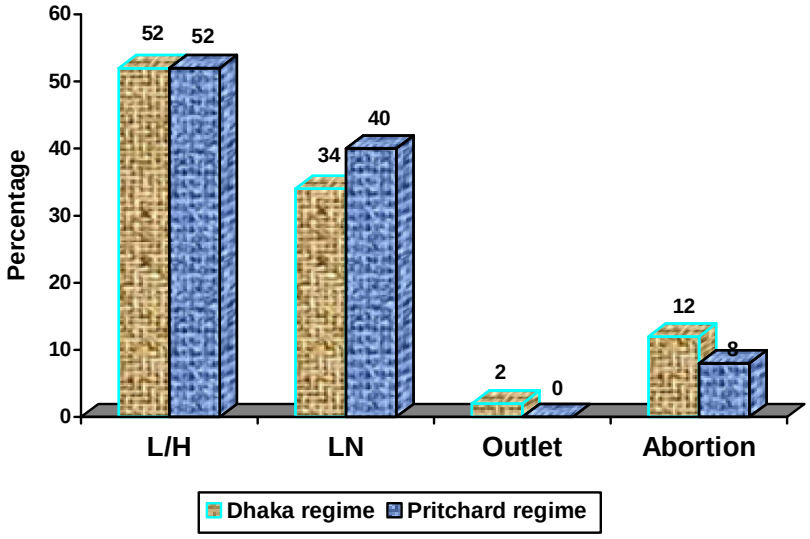
MODE OF DELIVERY

MODE OF DELIVERY	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
LSCS / Hysterotomy	26	52.0%	26	52.0%
Labour natural	17	34.0%	20	40.0%
Outlet forceps	1	2%	0	0%
Induced Abortion /spontaneous expulsion	6	12.0%	4	8%
Total	50	100.00%	50	100.00%

In patients treated with Dhaka regimen 26 underwent LSCS / Hysterotomy.17 delivered by labour natural. For 1 patient outlet forceps applied for fetaldistress. There were 6 induced abortions/ spontaneous expulsion. In patients treated with Pritchard 26 were underwent LSCS ,20 delivered labor naturally.

FIG : 10

MODE OF DELIVERY



L - LSCS

H - Hysterotomy

LN – Labour Natural

TABLE : 11

METHOD OF INDUCTION FOR VAGINAL DELIVERY

METHOD OF INDUCTION	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
PGE2 Gel	8	16.0%	8	16.0%
Oxytocin	12	24.0%	11	22.0%
ARM/Oxytocin augmentation	4	8.0%	5	10.0%
Total	24	48.00%	24	48.00%

In this study 48 patients delivered vaginally. In Dhaka regime 8 patients were induced with PGE2 gel and 12 patients were induced with oxytocin. Four patient's labor were augmented with artificial rupture of membrane followed by oxytocin infusion. In Pritchard regime, PGE2 gel and Oxytocin were used for 8 and 11 patients respectively.

FIG : 11

METHOD OF INDUCTION FOR VAGINAL DELIVERY

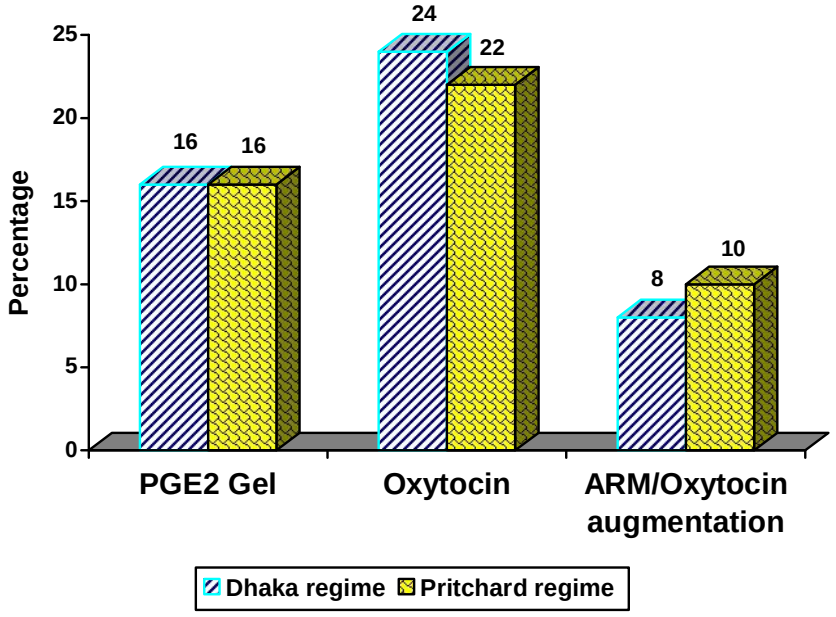


TABLE : 12

SEIZURE TO DELIVERY INTERVAL IN AP ECLAMPSIA

SEIZURE TO DELIVERY INTERVAL(Hr)	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
2	21	42.0%	18	36.0%
2 to 6	20	40.0%	19	38.0%
More than 6 Hrs	1	2.0%	4	8.0%
Total	42	84%	41	82.00%

There were 83 antepartum eclampsia cases in this study.

Out of 42 antepartum eclampsia patients treated with Dhaka regime,21 delivered within 2 hours.20 delivered within 2 to 6 hours. Only 1 delivered after 6 hours.

Out of 41 antepartum eclampsia patients 18 delivered within 2 hours. 19 delivered between 2 to 6 hours.4 patients took more than 6 hours.

FIG : 12

SEIZURE TO DELIVERY INTERVAL IN AP ECLAMPSIA

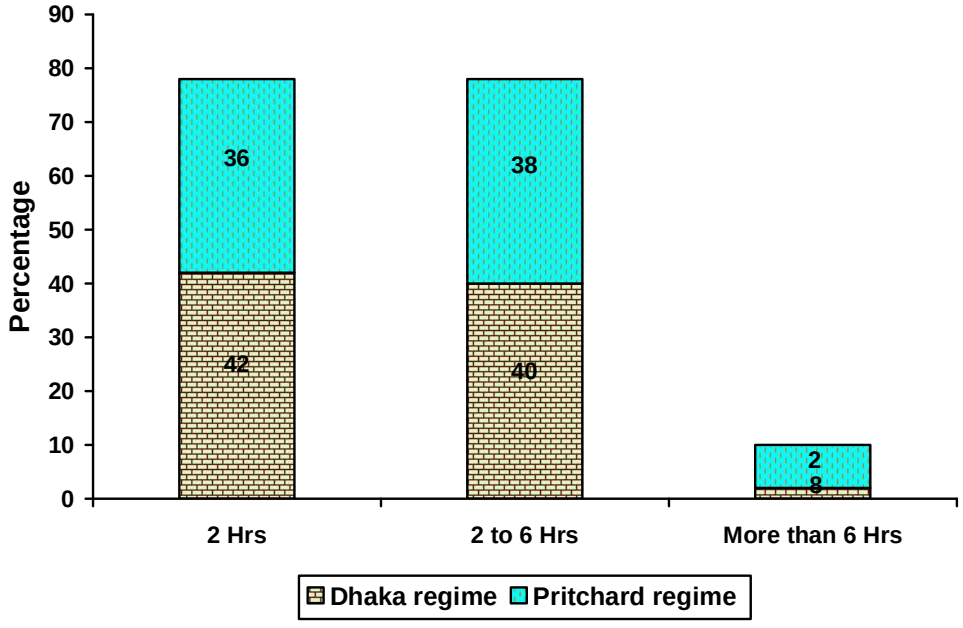


TABLE : 13

RECURRENCE OF FITS

Sl. No	Fits	Dhaka regimen		Pritchard regimen	
		(n=50)	(100%)	(n=50)	(100%)
1	Nil	49	98%	49	98%
2	Recurrence of fits	1	2%	1.0	2%

One patient from each regime had recurrence of fits. The patient in the Dhaka regime developed another convulsion after 3 hours of loading dose. One patient who developed recurrence of fit in Pritchard regime had that convulsion 2 hours after loading dose.

FIG : 13

RECURRENCE OF FITS

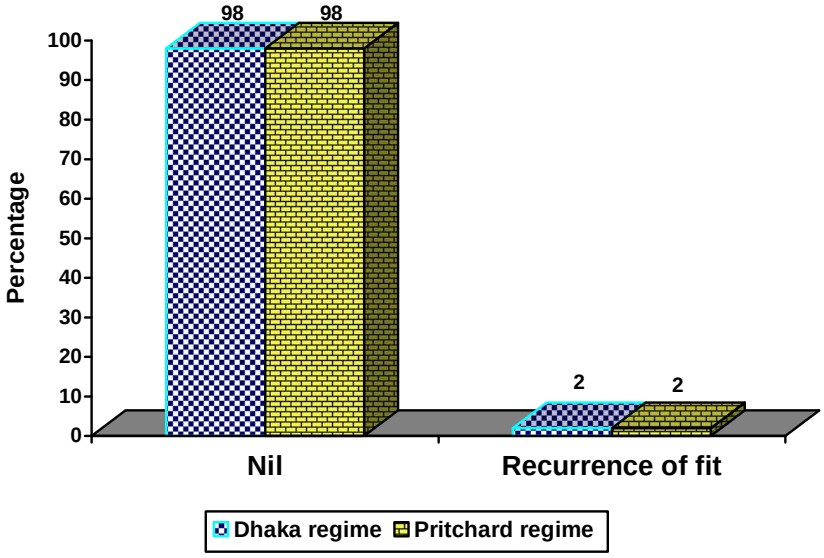


TABLE : 14

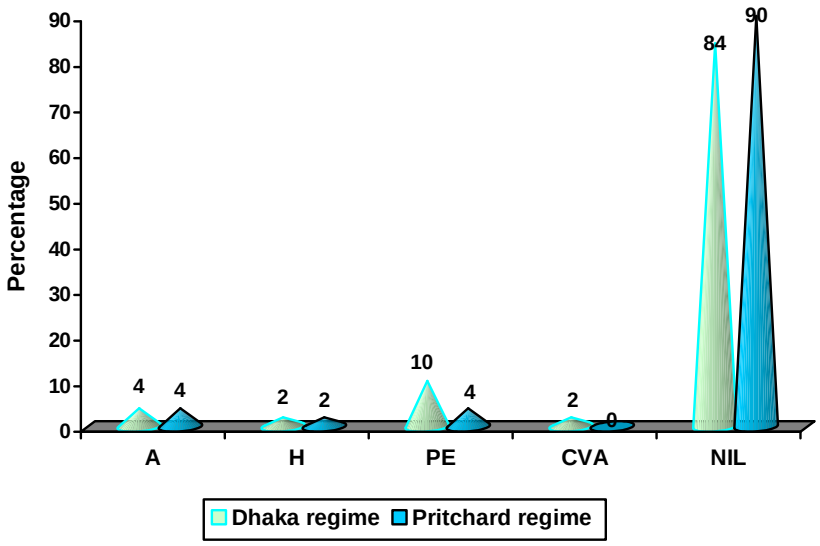
MATERNAL COMPLICATIONS

	Dhaka regime		**Pritchard regime**	
	(n=50)	**(100%)**	**(n=50)**	**(100%)**
Abruption	2	4.0%	2	4.0%
HELLP	1	2.0%	1	2.0%
Pulmonary Edema	5	10.0%	2	4.0%
Cerebro vascular accident	1	2.0%	-	0%
Nil	41	82.0%	45	90.0%
Total	50	100.00%	50	100.00%

In Dhaka regimen 2 patients had abruption,1 had HELLP and 5 had pulmonary edema.One patient developed occipital lobe infarct. In Pritchard regime 2 had abruption. 1 patient had HELLP and 2 had pulmonary edema.

FIG : 14

MATERNAL COMPLICATIONS



A –Abruption

H - HELLP

PE - Pulmonary edema

TABLE : 15

MATERNAL OUTCOME

MATERNAL OUTCOME	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Alive	49	98.0%	49	98.0%
Death	1	2.0%	1	2.0%
Total	50	100.00%	50	100.00%

In this study, out of the 100 patients, 98 were discharged from the hospital without any sequlae. 2 patients died due to complication of eclampsia.

FIG : 15

MATERNAL OUTCOME

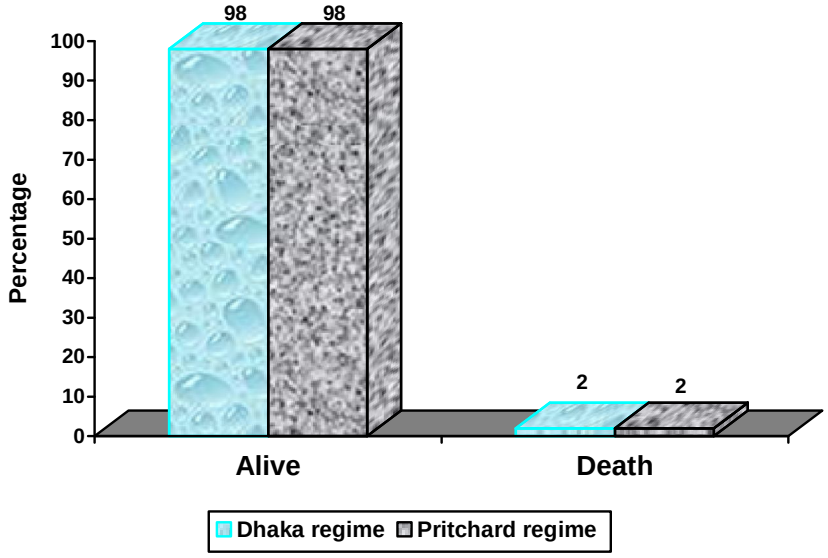


TABLE : 16

BIRTH WEIGHT

BIRTH WEIGHT	Dhaka regime		Pritchard regime	
	(n=50)	(100%)	(n=50)	(100%)
Less 1 kg	6	12.0%	4	8.0%
1 to 2kg	22	44.0%	29	58.0%
more than 2kg	22	44.0%	17	34.0%
Total	50	100.00%	50	100.00%

In Dhaka regimen the mean birth weight of the babies was 1.9332. In Pritchard regimen the mean birth weight of babies was 1.8410. The p value is 0.548

FIG : 16

BIRTH WEIGHT

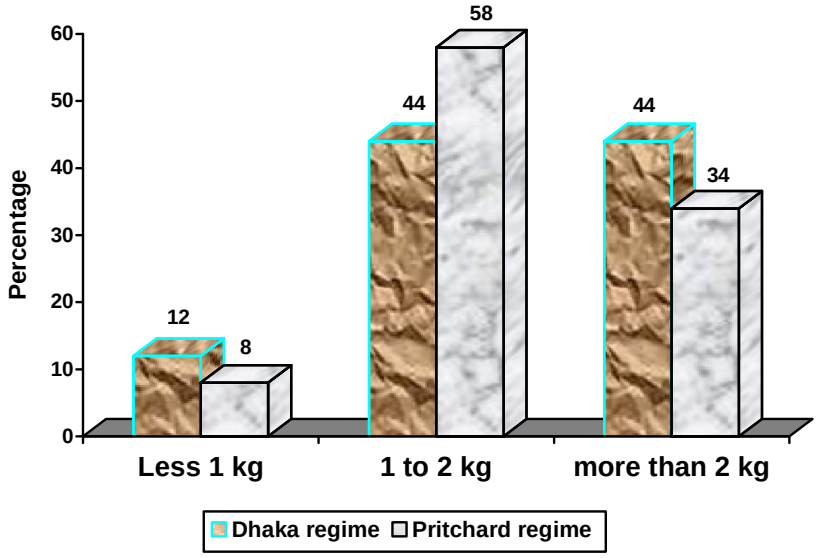


TABLE : 17

PERINATAL OUTCOME

Sl.no	Perinatal outcome	Dhaka regimen		Pritchard regimen	
		(n=50)	(100%)	(n=50)	(100%)
1	Alive	40	80.0%	37	74.0%
3	Perinatal death	10	20.0%	13	26%

In patients treated under Dhaka regimen 40 babies born alive. 10 died perinatally. In patients treated under Pritchard regimen 37 babies were alive and 13 died perinatally.

FIG : 17

PERINATAL OUTCOME

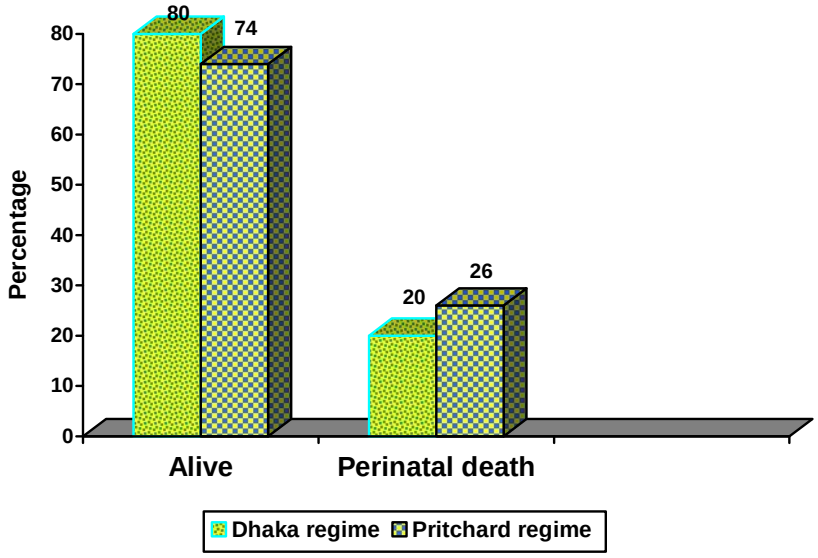


TABLE : 18

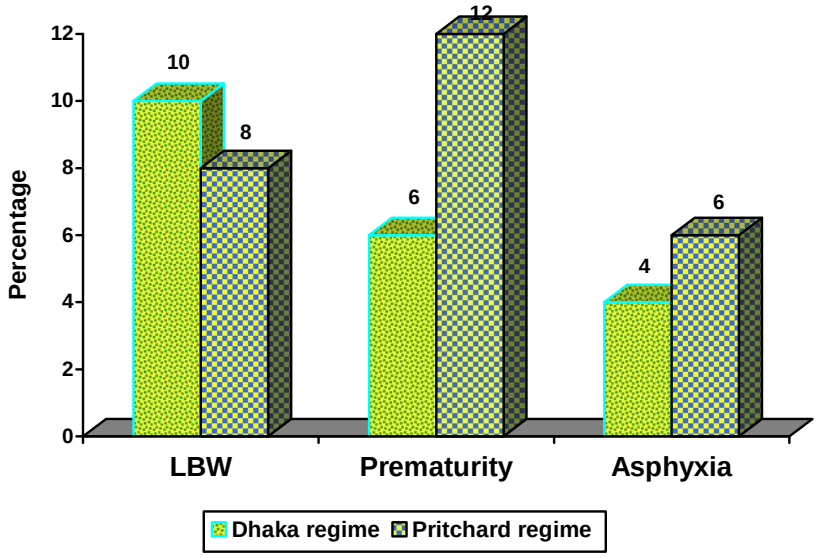
CAUSE OF PERINATAL DEATH

Sl.no	Cause of perinatal death	Dhaka regimen		Pritchard regimen	
		(n=50)	(100%)	(n=50)	(100%)
1	Low birth weight	5	10.0%	4	8.0%
2	Prematurity	3	6.0%	6	12%
3	Asphyxia	2	4.0%	3	6%

Among the 10 perinatal death in Dhaka regime patients, Low birth weight (10%), Pre maturity (6%) and Asphyxia (4%) were the cause of perinatal mortality. In Pritchard regime the above said causes contributed to 8%,12% and 6% respectively.

FIG : 18

CAUSE OF PERINATAL DEATH



DISCUSSION

The Maternal Morbidity is very much decreased with the control of eclamptic fits. $MgSO^4$ is an anticonvulsant drug used in the treatment of Antepartuum, intrapartum, postpartum eclampsia which has a wider margin of safety and rapid onset of action. Although rare the toxicity symptoms of magnesium toxicity can be corrected by antidote calcium gluconate which is readily available. Magnesium sulphate act as an ideal anti convulsant drug in the control of eclamptic fits. The Collaborative Eclampsia Trial has provided ample evidence that compared with other anti convulsants like Diazepam, phenytoin. Also the additional doses of $MgSO_4$ in the form of maintenance dose is not associated with any side effects to mother and her fetus.

Magnetic resonance angiographic imaging & computed tomographic scan has proved that vasospasm in the blood vessels of brain and thereby ischemia, which is the basic pathogenic mechanism responsible for the eclampsia and it is reversed by $MgSO_4$. Magnesium exhibit an inhibitory action on the cortical discharge from brain and oppose the glutamate N-methyl D- aspartate receptor, an excitatory receptor.

$MgSO_4$ adminsitrated also inhibit acetyl choline release at motor end plate level This is because of hypocalcemia caused by $mgso_4$ (Argany[45] and mejia –Mantilla, 2006)[33]

In this present study loading dose of $MgSO_4$ totally 10gms and maintenance dose of 2.5 gm $MgSO_4$ 4 hourly given as Intra muscular injection is given. It is just over half the dose of pritchard regimen.

AGE DISTRIBUTION:

In this present study, 47% of patients were in the age group of 21-25 yrs and for 25% the age was below 20. In 1989, a study conducted in N.W.M. hospital, Bombay reported that 56.8% were between 21-29 years, 40.5% were under 20 years and 2.7% were above 30 years. This present study is comparable to Lolkand et al (1997) study which reported that 40.7% eclamptic patients were under 20 years.

In this present study, the mean age of the patients studied under Dhaka regimen was 23.8 years and the mean age in Pritchard regimen was –23.26 years. Katz et al[46] (2000)study in sacred Heart Medical Center, the mean age of all eclampsia cases was 22 years.

PARITY DISTRIBUTION:

In this present study, among the 100 patients, 61% of patients were primi and 39% were multi. There were 64.9% primi and 35.1% were multi in a study conducted at N.W.H Hospital, Bombay.(1989) In Collaborative Eclampsia Trial[47] (1995) primi were 64% and multi were 36%. In both these studies, parity distribution is comparable to the present study.

GESTATIONAL AGE

In this study, in Dhaka regimen, the mean gestational age was –34.91 weeks and in Pritchard regimen the mean gestational age was35.06 weeks.

35% of the patients gestational age was more than 37 weeks. Of these patients 18% were treated under Dhaka regime and 17% were treated under Pritchard regime. In this present study 65% of patients had gestational age less than 37 weeks . In Collaborative Eclampsia Trial Group (1995) study, gestational age less than 34 weeks was 39.5% and gestational age between 34-36 weeks were 25.5% .Thirty three percent (33%) cases were at term gestation. Gestational age of the patients in the present study is comparable with collaborative eclampsia trial.

DIASTOLIC BLOOD PRESSURE

In this present study, the diastolic blood pressure range was 90- 110 and above. 54% patients who developed eclampsia had diastolic blood pressure between 90-110 mmHg. 37% of patients had severe preeclampsia with the diastolic blood pressure above 110 mmHg. And 9 % of the patients were normotensive. In Collaborative Eclampsia Trial group study (1995), patients with diastolic BP of more than 110 mm Hg were 53% .

RECURRENCE OF ECLAMPLSIA

In this study, the recurrence of eclamptic fits was 2 % .Reccurence of convulsions occurred both in Dhaka regime (one patient) and Pritchard regime (one patient). Collaborative Eclampsia Trial Group (1995) extensively studied 1687 women with eclampsia, They compared the efficacy of $MgSO_4$ with other anti convulsants namely Diazepam and Phenytoin. They showed 9.7% recurrence of seizures in the $MgSO_4$ regime.

Begum et al (1998) studied 65 eclamptic patients with Low dose MgSO4 (Dhaka regime). 1.5% of patients had recurrent convulsions. Bangal[48] et al (2009) studied 50 patients with eclampsia and who were treated with 4g of 20% $MgSO_4$ intravenously followed by 2gm of 50% $MgSO_4$ intramuscular every 4 hours and reported 6% recurrence of fits.And they shifted to Pritchard regime for these patients.

Bissallah[37] A Ekele et al (2009) studied 121 patients with eclampsia and treated with Ultra short regime (Sokotto regime). In ultra short regime loading dose of 4g of 20% $MgSO_4$ intravenously followed by 10gm of 50% $MgSO_4$ intramuscularly given. No maintenance dose was given for those patients. 7.4 % of patients developed recurrence seizures.

MODE OF DELIVERY

In this present study, under Dhaka regime 26 patients (52%) were delivered by Caesarean section/ hysterotomy. Remaining 24 patients (48%) delivered vaginally. 26 patients (52%) under Pritchard regime were delivered by Caesarean section. 24 patients (48%) delivered vaginally.

This observation in the mode of delivery is in contrast with Alexander and colleagues (1999)who induced labor in 50% cases. 15% patients responded and delivered vaginally. 35% of patients underwent caesarean section because of failed induction.

MATERNAL MORTALITY

In this present study, 2 patients (2%) died due to complication of eclampsia. One patient in Dhaka regime died of intra cranial haemorrhage. Since the patient's general condition was not conducive for mobilizing the patient, CT scan was not done. One patient in Pritchard regime died of pulmonary edema.

The patient admitted with the history of convulsion and admitted in a state of unconsciousness with pulmonary edema.

In Collaborative Eclampsia Trial ,the maternal mortality was 3.8% when treated with Magnesium sulphate. Bissallah A Ekele et al (2009) reported 12 maternal deaths in 121 patients (9.9%).

Shika sheth et al (2010)[49] studied 66 eclampsia patients and they categorized them into three groups. The patients were treated with Standard Pritchard regime, single loading dose regime and low dose MgSO4 regime respectively. Maternal mortality was 7.6% in Pritchard regime and 5% in single loading dose. There was no maternal mortality in patients treated with low dose regime.

MATERNAL MORBIDITY:

In this present study, the complication of eclampsia observed in patients treated under Dhaka regime were abruption (4%), HELLP (2%), pulmonary edema (10%) and cerebro vascular accidents (2%). In patients treated under Pritchard regime the incidence of abruption and HELLP were similar as in Dhaka regime . 4% of patients under Pritchard had pulmonary edema. The maternal morbidity between Dhaka regime and Pritchard regime was statistically significant.($p < 0.05$)

Shika seth et al (2010) reported pulmonary edema in patients treated with Pritchard regime(3.8%) and with single loading dose (5%). Cortical blindness (5%) reported in patients treated with low dose $MgSO_4$. This cortical blindness complication also observed in our present study in a patient treated under Dhaka regime.

According to Knight, United Kingdom Obstetric Surveillance system (UKOSS) in 2007 out of 214 eclamptic women 5 women had cerebral haemorrhage.

PERINATAL OUTCOME

In this present study, among the 100 patients 77 delivered alive babies.Perinatal death occurred in 23 babies.In Dhaka regime 80% babies were alive and remaining 20 % died in the perinatal period In Pritchard regime, there was 74% of alive birth and 13% of perinatal death. Shika seth et al (2010) reported 69% live birth , 31% perinatal death in patients treated under Pritchard regime. In the same study, they reported 75% live birth and 25% perinatal death in patients treated under low dose regime. In single loadaing dose regime there was 65% of live birth and 35% of perinatal death.

Gortzaek-Uzen[50] and associates (2005) pointed out that MgSO4 will cross placenta and the concentration of Mg in liquor is directly proportional to the time of Mg injection.But neonatal depression occurs only in cases of magnesium toxicity. None of our babies developed neonatal depression.

	Present study		Shika seth et al	Bangal et al	Begum et al	Bissallah A Ekele et al
	Dhaka	Pritchard				
Recurrence of fits	2%	2%	8%	6%	1.5%	7.4%
Maternal mortality	2%	2%	7.6%	0%	0%	9.9%
Maternal morbidity	18%	10%	3.8%	-	-	-
Perinatal death	20%	13%	31%	-	-	

This present study compared the efficacy of low dose $MgSO_4$ with standard Pritchard regime. This study showed that recurence of fits, maternal mortality, maternal morbidity and perinatal outcome were not statistically significant between Dhaka regime and Pritchard regime. Recurrence of fits in this study is comparable with the Begum et al (1998) (2% vs 1.5%). The recurrence of fit is found to be higher in Shika seth et al, Bangal et al and Bissallah A Ekele et al studies.(8%, 6% and 7.4%)

The maternal mortality was nil in Begum et al and in Bangal et al studies. The present study observed 2% maternal mortality. This is less than the reported mortality of Shika seth et al (7.6%) and Bissallah A Ekele et al (9.9%).

The maternal morbidity was found to be higher than in Shika Seth study (18% vs 3. 8%).

SUMMARY

In this present study 100 patients of eclamptic women irrespective of the type of eclampsia (Antepartum/ Intrapartum/ Postpartum) were included. 50 patients were treated under Dhaka regimen. Another 50 eclamptic patients were treated under Pritchard regime. And the two group of patients were observed for the recurrence of fits, maternal morbidity, mortality as well as for perinatal outcome. Thus the safety and efficacy of low dose magnesium sulphate was compared with Pritchard's regimen. With respect to seizure recurrence, 2 patients each one from Dhaka and Pritchard regime had recurrent fit. The recurrent seizure patients were treated with additional dose of 2g of 20% $MgSO_4$ Intravenously. There were no symptoms of Magnesium toxicity in both the patients.

In both study and control groups, totally 98 patients discharged in healthy state. Two patients died due to complication. Perinatal outcome of patients treated in the two different regime are comparable. The perinatal outcome does not differ significantly in both Dhaka and Pritchard regimen groups.

CONCLUSION

- Most of the Indian women considered to be of low to normal BMI need only low dose magnesium sulphate which is sufficient for them
- Efficacy of low dose $MgSO_4$ regime in controlling the convulsions, and its effect on maternal and perinatal outcome is equivalent to standard Pritchard regime.
- Low dose magnesium sulphate is equally good enough to control eclampsia, thereby preventing magnesium toxicity though it is rare
- Poor manpower and waste of time in monitoring the Mg toxicity symptoms can be avoided in low dose $MgSO_4$.

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PROFORMA

Name:

Age:

IP No:

Socio economic status:

Date & Time of admission:

Date & Time of Delivery:

Date of discharge:

Obstetric code:

LMP:

EDD:

GA:

Booked/Unbooked:

Referred from:

H/O PI:

G.C –conscious

postictal confusion

unconscious

No of convulsion before admission

H/O of imminent symptoms

Pedal edema

Obstetric H/O

Past H/O

Co morbid conditions

O/E

Conscious/unconscious/drowsy

Oriented

Pallor

Pedal edema

BMI

BP

RR

CVS

RS

DTR

P/A

P/V

Bishop’s score

Investigation

Urine

- Alb
- Sugar

Deposits

24 hours protein

Complete haemogram

BT

CT

CRT

BLOOD SUGAR

BLOOD UREA

SR.Creatinine

Sr.Electrolytes

Peripheral Smear

USG

Fundus

Outcome-

Maternal Outcome

Mode of Delivery-Vaginal/

LSCS/Hysterotomy

Abortion

Regimen of $MgSO_4$ given

No of convulsion before starting $MgSO_4$

No of convulsion recurrent after starting $MgSO_4$

Any sign of Magnesium toxicity

Complication during hospital stay

General condition of mother during discharge-healthy/died

Fetal outcome

- alive
- dead

APGAR score

Birth weight

Condition of newborn during discharge

Healthy/died

Cause of neonatal death

Sl.No.	Name	Age	IP No	Obstetric Formula	Booking Status	Gest Age [WKS]	BMI	No.of Seizures Bef Adm	State of consciousness	Blood Pressure (mm Hg)		Regime given	AP/IP/PP	Method of Induction	Mode of Delivery	Complication in IP /PP	Admission - delivery / delivery Seizure interval	Maternal outcome				Perinatal Outcome	
										Systolic	Diastolic							Recurrence of fits	Magnesium Toxicity sign	Dose deferral	Condition of Discharge	BWT	Condition
1	Rajathy	20	159433	Primi	2	26	N	1	1	160	100	D	1	1	A		6	_	_	_	1	1.15	2
2	Priya	26	158289	Primi	2	34	N	1	1	170	100	D	1	1	S		6	_	_	_	1	0.95	2
3	Madhuri	22	162623	Primi	1	38	N	1	1	160	110	P	1	1	V	A	6	_	_	_	1	1.23	1
4	Kasthuri	22	165029	G2PL1	1	36	N	1	1	170	100	D	1	0	S		1	_	_	_	1	2	1
5	Senthamarai	26	167276	Primi	1	37	N	1	1	160	110	P	1	2	V		3	_	_	_	1	2.7	1
6	Christura	23	168779	Primi	1	38	N	2	1	150	110	P	1	2	V		4	_	_	_	1	2.3	1
7	Chitravalli	23	167568	G2PL1	1	34	N	1	1	170	100	D	1	2	V		4	_	_	_	1	1.8	2
8	Shobha	21	168581	Primi	1	41	N	1	1	160	100	D	2	2	O		1	_	_	_	1	2.8	1
9	Vasuki	24	168535	Primi	1	28	N	2	2	160	90	P	1	3	V		2	_	_	_	1	1	1
10	Amudha	22	169934	Primi	2	35	N	1	1	150	110	D	1	2	V		4	_	_	_	1	2	1
11	Latha	30	173396	G2PL1	2	31	N	2	1	130	90	D	1	2	V		2	_	_	_	1	0.9	1
12	Rajavalli	24	174543	G2PL1	1	39	N	1	1	150	110	P	1	0	S	A	1	_	_	_	1	2.75	1
13	Jaseema	22	174454	G2A1	1	38	N	1	1	160	110	D	1	1	S		4	_	_	_	1	2.2	1
14	Maheswari	21	174326	Primi	1	37	N	1	1	150	100	P	1	3	V		3	_	_	_	1	2	1
15	Saral	30	174637	P1L1	1	26	N	1	1	160	120	D	3	0	A		1	_	_	_	1	2.3	1
16	Rashidha	19	176673	Primi	1	44	N	1	1	130	100	D	2	3	S		30	_	_	_	1	2.8	1
17	Mahalakshmi	25	177086	Primi	1	35	N	1	1	170	110	D	1	3	V		1	_	_	_	1	1.5	2
18	Sathya	23	177125	G2PL1	2	32	N	1	1	150	100	P	1	3	V		6	_	_	_	1	1.2	2
19	Sunithra	22	177501	Primi	1	39	N	1	1	150	110	D	1	0	S		1	_	_	_	1	2.75	1
20	Deepa	20	177535	Primi	2	29	N	1	1	160	110	P	1	2	V		6	_	_	_	1	1.25	1

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										Systolic	Diastolic							Recurrence of fits	Magnesium Toxicity sign	Dose deferral	Condition of Discharge	BWT	Condition
21	Meenakshi	28	177320	G2PL1	1	37	N	1	1	150	100	P	1	0	S		1	_	_	_	1	1	2
22	Senthamarai	35	177857	G2P10	1	24	N	1	1	160	110	D	1	1	S		6	_	_	_	1	0.6	1
23	Nisha	27	177925	G2PL1	1	41	N	1	1	180	130	D	1	1	V		5	_	_	_	1	2.7	1
24	Muthu	22	178060	P1L1	2	39	N	1	1	150	90	P	1	0	S		1	_	_	_	1	3.5	1
25	Muthulakshm	27	178152	P2L2	1	38	N	1	1	170	110	P	1	0	V		1	_	_	_	1	2.5	1
26	Arultherasa	32	178152	G4P3L3	2	38	N	1	1	170	100	D	1	3	S		6	_	_	_	1	1.9	1
27	Revathy	21	178343	Primi	1	38	N	1	1	160	110	D	1	1	V		6	_	_	_	1	3	1
28	Kalaiarasi	23	178329	P1L2	2	31	N	1	1	150	100	D	1	0	S		1	_	_	_	1	1.75	2
29	Joice	23	178827	Primi	2	28	N	10	3	180	110	P	1	1	A		3	1	_	_	1	1	2
30	Meena	22	179094	Primi	1	36	N	1	1	150	100	D	1	3	S		2	_	_	_	1	2.15	1
31	Sudha	30	179222	Primi	2	32	N	1	1	150	100	P	1	3	S		1	_	_	_	1	1.5	1
32	Viji	24	179141	Primi	1	41	N	2	1	160	110	D	1	1	V		2	1	_	_	1	2.4	1
33	Rathna	19	179501	G2A1	1	32	N	1	2	110	80	P	1	1	V		8	_	_	_	1	1.5	1
34	Ilaveni	14	180201	Primi	2	32	L	6	1	140	90	D	1	2	V		4	_	_	_	1	1.3	1
35	Banupriya	19	180745	Primi	1	33	N	1	1	170	110	D	1	1	V		6	_	_	_	1	1.2	1
36	Sheelarani	30	180783	G2PL1	1	36	H	1	1	130	90	P	3	0	S		1	_	_	_	1	2.5	1
37	Nalini	26	180840	Primi	2	32	N	1	1	150	100	P	1	3	V		2	_	_	_	1	0.8	1
38	Latha	22	181318	G2A1	2	37	N	2	1	160	110	P	1	1	V		6	_	_	_	1	0.75	1
39	Alageswari	20	183411	Primi	1	39	N	4	1	150	90	D	1	1	V		6	_	_	_	1	3	1
40	Umashanthi	30	183714	G2P4	2	38	N	2	1	130	90	D	1	1	V	A	3	_	_	_	1	0.6	1

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										Systolic	Diastolic							Recurrence of fits	Magnesium Toxicity sign	Dose deferral	Condition of Discharge	BWT	Condition
41	Jeyapriya	24	184151	Primi	1	34	H	1	1	160	110	P	1	0	S		2	_	_	_	1	1.9	1
42	Revathy	26	184233	Primi	1	30	N	1	1	170	110	P	1	2	S		5	_	_	_	1	1.8	1
43	Selvarani	20	184398	G2A1	1	38	H	1	1	150	100	P	1	2	S		1	_	_	_	1	1.47	1
44	Kanagavalli	22	185706	P1L1	1	36	O	3	1	160	100	P	3	0	S		7	_	_	_	1	3	1
45	Uma	23	185103	Primi	2	28	N	4	1	140	100	D	3	2	V		3	_	_	_	1	0.7	2
46	Nithya	20	186514	Primi	1	37	N	1	3	160	100	D	3	0	V		1	_	_	_	1	2.1	1
47	Nadhiya	21	182479	Primi	1	38	N	2	1	140	100	D	1	0	S		1	_	_	_	1	2.5	1
48	Mahalakshmi	21	187075	Primi	2	36	N	2	1	130	80	D	1	0	S		1	_	_	_	1	1.7	1
49	Jeromiya	22	187121	Primi	1	36	N	2	3	180	130	P	1	0	S		1	_	_	_	1	1.25	2
50	Balamani	21	187904	Primi	2	37	N	1	1	158	100	D	1	0	S		1	_	_	_	2	1.55	3
51	Prabha	22	189114	Primi	1	39	N	2	1	160	110	D	1	0	S		3	_	_	_	1	3.3	1
52	Ilaveni	14	180201	Primi	2	37	L	2	2	160	100	D	1	1	V		6	_	_	_	1	1.3	2
53	Amudha	30	189305	P6L5	2	37	L	1	1	120	70	D	3	0	V		48	_	_	_	1	2.3	1
54	Padmini	20	188023	Primi	1	39	N	1	1	130	100	P	1	0	S		2	_	_	_	1	3.1	1
55	Indhumathy	19	189753	Primi	1	36	N	1	1	160	100	D	1	0	V	H	1	_	_	_	1	2.2	2
56	Leo jancy ma	25	189473	G2A1	2	32	N	2	1	150	110	P	1	0	S		1	_	_	_	1	1.2	2
57	Vasugi	25	190357	G2PL1	1	36	N	1	1	130	90	D	3	2	V		4	_	_	_	1	2.5	1
58	Malathy	25	190697	Primi	1	37	N	2	1	160	90	D	1	0	S		1	_	_	_	1	1.2	1
59	Dhanalakshm	27	191121	G3P2L2	2	39	N	6	1	150	100	P	1	0	V		1	_	_	_	1	1.3	1
60	Nadhiya	22	191944	G2PL1	2	34	N	1	1	140	100	D	1	2	V		5	_	_	_	1	2	1

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										Systolic	Diastolic							Recurrence of fits	Magnesium Toxicity sign	Dose deferral	Condition of Discharge	BWT	Condition
61	Sathyabama	20	192051	G3P2L2	1	37	N	1	1	160	100	P	3	3	S		1	_	_	_	1	2.5	1
62	Karpagam	26	192258	G2P1L0	1	37	N	3	1	160	110	P	1	0	S		1	_	_	_	1	2.3	1
63	Parimala	21	192364	Primi	1	35	H	1	1	140	100	P	1	1	S		4	_	_	_	1	1	1
64	Malathy	21	192371	Primi	1	37	N	3	1	140	100	D	1	_	S		1	_	_	_	1	2.75	1
65	Saranya	19	193084	Primi	1	34	N	2	1	160	110	P	3	2	V		4	_	_	_	1	2	1
66	Dhavamani	27	193516	Primi	1	35	N	2	1	130	80	P	3	2	V	H	2	_	_	_	1	2	2
67	UshaNandhin	28	194284	G2PL1	1	34	N	1	1	120	80	D	3	_	S		1.2	_	_	_	1	1.75	1
68	Revathy	22	194275	Primi	1	32	N	2	1	150	110	P	1	_	S		3	_	_	_	1	1.3	1
69	Rani	20	194350	Primi	2	38	N	3	1	160	100	P	1	_	S		1	_	_	_	1	14	1
70	Jeyanthy	24	197044	Primi	2	28	N	2	1	130	90	P	1	1	A		12	_	_	_	1	1.25	2
71	Perumayi	24	197020	G2P11	2	37	L	0	1	200	140	D	2	0	V		15	_	_	_	1	2.54	2
72	Mariammal	18	196608	Primi	2	34	N	1	1	130	90	P	1	1	S		4	_	_	_	1	1.2	1
73	Vasanthi	35	196611	G2P14	1	37	N	0	1	130	90	D	2	0	S		15	_	_	_	1	3.2	1
74	Sudha	27	197264	G3A2	1	38	N	1	1	170	110	P	1	12	V		6	_	_	_	1	1.2	1
75	Vembu	23	196899	P1L1	2	38	N	1	1	150	90	P	3	0	S		1	_	_	_	1	3.8	1
76	Madhuramba	20	196871	Primi	2	32	N	1	1	200	110	D	1	0	S		1	_	_	_	1	1.3	1
77	Kayalvizhi	26	198330	Primi	1	38	N	1	2	170	130	D	1	1	S		5	_	_	_	1	2	1
78	Kalaivani	22	199472	G2PL1	1	34	N	3	1	140	90	D	1	0	S		1	_	_	_	1	1.3	1
79	Kannammal	20	200830	Primi	2	36	N	1	1	130	80	P	1	1	V		7	_	_	_	1	1.4	1
80	Ramya	20	202324	Primi	2	34	N	2	1	160	100	P	1	_	S		4	_	_	_	2	1.6	1

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										Systolic	Diastolic							Recurrence of fits	Magnesium Toxicity sign	Dose deferral	Condition of Discharge	BWT	Condition
81	Radhika	24	202341	Primi	1	33	N	2	1	100	70	P	1	1	V		7	_	_	_	1	1.6	1
82	Vaideki	18	203624	Primi	2	28	N	1	1	160	90	D	1	1	A		2	_	_	_	1	1	1
83	Jegadhamba	20	203741	G2A1	2	35	0	6	3	190	120	P	1	0	S	PE	1.5	1	_	_	1	1.8	1
84	Sangeetha	28	203950	G3P2L2	1	38	N	1	1	160	100	P	2	3	V		1.5	_	_	_	1	2.7	1
85	Nirosha	23	204083	G2A1	1	40	N	1	1	160	100	P	1	_	S		1	_	_	_	1	3 KG	1
86	Vembarasi	28	206262	Primi	1	38	N	1	1	140	90	D	1	2	V		4.5	_	_	_	2	2.7	1
87	Malaiyalatha	35	206876	G3A2	1	24	N	1	1	160	100	D	1	2	S		12	_	_	_	1	750 G	1
88	Radhika	27	206766	Primi	2	34	N	1	1	150	90	D	1	S	S		2	_	_	_	1	1.85	1
89	Sheelarani	22	209206	Primi	1	40	N	5	2	150	110	P	1	_	S		2	_	_	_	1	2.85	1
90	Rajathi	20	211372	Primi	1	26	N	3	1	200	110	P	1	1	V		6	_	_	_	1	500G	1
91	Meenatchi	24	212862	Primi	2	35	N	2	1	170	110	P	1	_	S		4	_	_	_	1	1.3	1
92	Chitra	23	213061	Primi	1	37	N	3	2	170	110	D	1	_	S		2	_	_	_	1	2.5 KG	1
93	Muthulakshm	22	214365	G3P1L1A1	2	38	N	1	1	90	60	P	1	1	V		5	_	_	_	1		2
94	Shanthi	35	216215	G4P3L3	2	37	N	3	1	160	110	D	1	1	V	A	3	_	_	_	1	1.41	1
95	Parameswari	19	219050	Primi	1	41	N	2	1	130	90	P	1	2	V	A	7	_	_	_	1	3.14	1
96	Priya	20	23088	Primi	2	34	L	4	2	150	110	D	1	_	S	CVA	1	1	_	_	2	1.8	1
97	Karthiga	24	230556	Primi	2	37	N	1	1	110	70	P	1	2	S		2	_	_	_	1	2.6	1
98	Jegadeeswar	27	230678	Primi	2	35	N	2	1	130	90	P	1	2	S		3	_	_	_	1	2.66	1
99	Rajalakshmi	23	280683	Primi	1	36	N	1	1	140	90	D	1	2	S		2	_	_	_	1	2.76	1
100	Vijayashanthi	20	233055	Primi	1	35	N	4	2	160	100	P	1	2	V		8	_	_	_	1	1.2	2